Andrew Orenstein, Esq. August 21, 2007 Page 32

- 130. Preston D.L. et al. Studies of mortality of atomic bomb survivors. Report 13: Solid cancer and noncancer diseased mortality: 1950-1997. Radiat Res, 160(4):381-407, October 2003.
- 131. Removal of Non-Friable Asbestos-Containing Floor Tile Using the Resilient Floor Covering Manufacturer "Recommended Work Practices for the Removal of Resilient Floor Covering," Micro-Analytics, Inc., June 10, 1992.
- Resilient Floor Covering Institute. Recommended Work Practices for Removal of Resilient Floor Coverings, January 1998.
- 133. Review and Analysis of Studies That Monitored Fiber Exposures During Maintenance of Asbestos-Containing Resilient Floor Tiles, ENVIRON Corporation, November 30, 1990.
- 134. Review and Analysis of Studies That Monitored Fiber Exposures During Removal of Asbestos-Containing Resilient Floor Covering Materials, ENVIRON Corporation, November 30, 1990.
- Risberg B. et al. Familial clustering of malignant mesothelioma. Cancer, 45:2422-2427, 1980.
- 136. Robinson C. et al. Mortality patterns, 1940-1975 among workers employed in an asbestos textile friction and packing products manufacturing facility. Circa 1979.
- 137. Rogers A.J. et al. Relationship between lung asbestos fiber type and concentration and relative risk of Mesothelioma. Cancer, 67:1912-1920, 1991.
- 138. Roggli V.L. The role of analytical SEM in the determination of causation in malignant mesothelioma. Ultrastructural Pathology, 30:31-35, 2006.
- 139. Roggli V.L. et al. Malignant mesothelioma and occupational exposure to asbestos: a clinicopathological correlation of 1445 cases. Ultrastructural Pathology, 26(2):55-65, MarApr 2002.
- Roggli V.L. et al. Tremolite and mesothelioma. Annals of Occupational Hygiene, 46(5):447-453, 2002.
- 141. Rohl A.N. et al. Asbestos exposure during brake lining maintenance and repair.Environmental Research, 12(1):110-28, August 1976.142. Rohl A.N. et al. Exposure to asbestos in the use of consumer spackling, arching.
- taping compounds. Science, 189:551-553, August 1975.

  143. Rubino G.F. et al. Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. British Journal of Industrial Medicine, 36(3):187-194, August, 1979.
- 144. Sakai K. et al. Asbestós and nonasbestos fiber content in lung tissue of Japanese patiel with malignant mesothelioma. Cancer, 73:1825-1835, 1994.

Ę

Andrew Orenstein, Esq. August 21, 2007 Page 33

- 145. Sanders C.L. Pleural mesothelioma in the rat following exposure to 239PuO2. Health Phys, 63(6):695-697, December 1992.
- Saracci R. and Simonato L. Familial malignant mesothelioma. Lancet, 358(9295):1813-1814, November 24, 2001.
- 147. Serio G. et al. Familial pleural mesothelioma with environmental asbestos exposure: losses of DNA sequences by comparative genomic hybridization (CGH). Histopathology, 45(6):643-645, December 2004.
- 148. Shilmikova N.S. et al. Cancer mortality risk among workers at the Mayak nuclear complex. Radiat Res, 159(6):787-798, June 2003.
- 149. Smith A.H. et al. Chrysotile asbestos is the main cause of pleural mesothelioma. American Journal of Industrial Medicine, 30:252-266, 1996.
- Spirtas R. et al. Malignant Mesothelioma: attributable risk of asbestos exposure.
   Occupational and Environmental Medicine, 51:804-811, 1994.
- 151. Spirtas R. et al. Malignant mesothelioma: attributable risk of asbestos exposure. Occup Environ Med, 51(12):804-811, December 1994.
- 152. Spirtas R. et al. Mesothelioma risk related to occupational or other asbestos exposure: preliminary results from a case control study. Society for Epidemiologic Research: Abstracts. American Journal of Epidemiology, 122:518, 1985.

. 5

-

- 153. Srebro S.H. et al. Asbestos-related disease associated with exposure to asbestiform Tremolite. American Journal of Industrial Medicine, 26:809-819, 1994.
- SRI International. Comparison Testing Monitoring for Airborne Asbestos Fibers: Sheet Vinyl Floor Covering, Wet Versus Dry Scraping, Final Report, SRI International Project 7988, November 1979.
- 155. SRI International. Monitoring for Airborne Asbestos Fibers: Vinyl Asbestos Floor Tile, Final Report, SRI International Project 7988, December 1979.
- 156. Stanton M.F. and Wrench C. Mechanisms of mesothelioma induction with asbestos and fibrous glass. Journal of the National Cancer Institute, 48:797-821, 1972.
- 157. Subramanian V. and Madhavan N. Asbestos problem in India. Lung Cancer, 49 Sur 1:S9-S12, July 2005.
- 158. Suzuki Y. et al. Asbestos fibers contributing to the induction of human malignant mesothelioma. Annals of the New York Academy of Science, 982:160-76 December 2002.
  - 159. Suzuki Y. et al. Asbestos tissue burden study on human malignant mesothelioma. Industrial Health, 39(2):150-160, April 2001.

Andrew Orenstein, Esq. August 21, 2007 Page 34

- Svorcan P. et al. Primary malignant mesothelioma of the peritoneum. Rom J Gastroenterol, 12(2):135-137, June 2003.
- 161. Teschke K. et al. Mesothelioma surveillance to locate sources of exposure to asbestos. Canadian Journal of Public Health, 88(3):163-168, May-June 1997.
- Teta M.J. et al. Mesothelioma in Connecticut, 1955-1977. Journal of Occupational Medicine, 25(10):749-756, October 1983.
- 163. Teta M.J. et al. Therapeutic radiation for lymphoma: risk of malignant mesothelioma. Cancer, 109(7):1432-1438, April 1, 2007.
- 164. Thomas H.F. et al. Further follow up study of workers from an asbestos factory. British Journal of Industrial Medicine, 39:273-276, 1982.
  - Tossavainen A. et al. Amphibole fibres in Chinese chrysotile asbestos. Annals of Occupational Hygiene, 45(2):145-52, March 2001.
- Tossavainen A. et al. Pulmonary mineral fibers after occupational and Environmental Exposure to Asbestos in the Russian Chrysotile Industry. American Journal of Industrial Medicine, 37:327-333, 2000.
- 167. Travis L.B. et al. Second cancers among 40576 testicular cancer patients: focus on long-term survivors. J Natl Cancer Inst, 97(18):1354-1365, 2005.
- 168. Tward J.D. et al. The risk of secondary malignancies over 30 years after the treatment of non-Hodgkin lymphoma. Cancer, 107(1):108-115, July 1, 2006.
- 169. U.S. Environmental Protection Agency. Asbestos-in-Buildings Technical Bulletin: Use of Asbestos-Containing Friable Materials and Vinyl-Asbestos Floor Tiles in Public and Commercial Buildings, U.S. Environmental Protection Agency, TS-798:1-4, 1984.
- 170. Ulvestad B. et al. Cancer incidence among workers in the asbestos-cement producing industry in Norway. Scand J Work Environ Health, 28(6):411-417, 2002.
- 171. Vinyl Asbestos Floor Tile Study, Routine Buffing and Stripping Operations for WRC-TV Washington, A.F. Meyer and Associates, Inc., November 6, 1989.
- 172. Wagner J.C. et al. Correlation between lung fibre content and disease in East London asbestos factory workers. IARC Sci Publ, (90):444-448, 1989.
  - 173. Wagner J.C. et al. Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. British Journal of Industrial Medicine, 17:260-271, 1960
- 174. Warnock M.L. Lung asbestos burden in shipyard and construction workers with mesothelioma: comparison and burdens in subjects with asbestosis or lung cancer. Environmental Research, 50(1):68-85, October 1989.

Andrew Orenstein, Esq. August 21, 2007 Page 35

- 175. Weiss et al. Mortality of a cohort exposed to chrysotile asbestos. Journal of Occupational Medicine, 19(11):737-740, November 1977.
- 176. Welch L.S. et al. Asbestos and peritoneal mesothelioma among college-educated men. Int J Occup Environ Health, 11:254-258, 2005.
- 177. Williams M.G. and Crossman R.N. Asbestos release during removal of resilient floor covering materials by recommended work practices of the Resilient Floor Covering Institute. Applied Occupational and Environmental Hygiene, 18(6):466-478, 2003.
- 178. Woitowitz H.J. et al. Mesothelioma among car mechanics. Annals of Occupational Hygiene, 38(4):635-638, 1994.
- 179. Wong I. Malignant mesothelioma and asbestos exposure among auto mechanics: appraisal of scientific evidence. Regulatory Toxicology and Pharmacology, 34:170-177, 2001.
- 180. Yan T.D. et al. Significance of lymph node metastasis in patients with diffuse malignant peritoneal mesothelioma. Eur J Surg Oncol, 32(9):948-953, November 2006.
- 181. Yano E. et al. Cancer mortality among workers exposed to amphibole-free chrysotile asbestos. American Journal of Epidemiology, 154(6):538-543, 2001.
- 182. Yarborough C.M. Chrysotile as a cause of mesothelioma: an assessment based on epidemiology. Critical Reviews in Toxicology, 36:165-187, 2006.

#### **EXHIBIT C**



August 22, 2007

# VIA OVERNIGHT MAIL

Cynthia Weiss Antonucci, Esq. Harris Beach, PLLC New York, NY 10005 100 Wall Street

Jack Nacht, Index No. 114274-06, New York Co. Re:

## Dear Ms. Antonucci:

Thank you for referring this case for medical review and analysis. The records indicate that this case involves a claim of mesothelioma. Specifically, it is claimed that Mr. Nacht was exposed to asbestos-containing floor tile over the many years that he owned a carpet and tile business and that exposure was causal for the development of his mesothelioma.

I am a licensed physician specializing in occupational and environmental medicine. I was trained in the field while serving as a medical officer with the National Institute for Occupational Safety & Health ("NIOSH"), an institute within the Centers for Disease Control and Prevention agencies. I was invited to serve on a National Academy of Sciences panel addressing causation this field. Occupational and environmental medicine is a sub-specialty of preventive medicine which deals with the recognition, assessment, control and prevention of potentially hazardous in an occupational health concern. I also have been invited to serve on a committee within the lectured widely in the field, including courses and lectures on causation determination. I have before various state legislatures, and in state and federal court proceedings. I have taught and ("CDC") of the United States Public Health Service. I also have trained with noted experts in workplace and community exposures. I have practiced in the field since 1979. As part of my experience and training in this field, I utilize the principals and practices of toxicology, epidemiology, industrial hygiene and occupational and environmental medicine, among other occupational and environmental health issues on several occasions before the U.S. Congress, Consumer Product's Safety Commission (CPSC), and other state and federal government Administration ("OSHA"), the U.S. Environmental Protection Agency ("EPA"), the U.S. related disciplines. I have consulted for NIOSH, the U.S. Occupational Safety & Health NIOSH National Occupational Research Agenda. I have provided expert testimony on

Innovation Way, Sulte 400

New York 125 Baylis Road, Sulte 120 Melville. New York 11747

Galthersburg, MD 20879-4145 22 Cessna Court

Maryland

Cynthia Weiss Antonucci, Esq. August 22, 2007

assessments of possible pneumoconioses and malignancies and potential exposure in individuals published and contributed to texts in the field. I also have contributed to various governmental epidemiological study of thousands of workers potentially exposed to various inorganic dusts including silica and asbestiform minerals. Throughout my career I have performed many reports and criteria for regulatory development. I currently am conducting a multi-year and groups.

the scientific and medical literature regarding exposure and health effects of asbestos and other inorganic dusts. I also have participated in the exposure analysis of various asbestos-containing employers with asbestos-exposed workers. I have routinely evaluated and continue to evaluate Specifically with regard to asbestos, I performed a health assessment of asbestos and asbestos substitutes for the CPSC. I conducted an epidemiologic study on a wide range of job duties at a facility where asbestos-containing materials were employed including insulation materials. I have designed and conducted medical monitoring and surveillance for various products, materials and environments along with my industrial hygiene staff.

development of mesothelioms and asbestos exposure are based on my training, experience, study Opinions offered regarding presence of alleged disease, exposure to possible risk factors, causation, alternative etiologies, warnings and scientific state of the art regarding the conclusions are also based on my review and evaluation of the various case materials including: and continuing assessment of the relevant scientific and medical literature. My opinions and

- Summions and Complaint
- Kentile's Answer to Complaint
- Plaintiff's Responses to Defendant's Fourth Amended Standard Set of Interrogatories and Request for Production of Documents
  - Social Security Administration Records
- Depositions:
- Jack Nacht, November 21, 2006, Vol I
- Jack Nacht, December 4, 2006, Vol II Jack Nacht, February 6, 2007, Vol III

0 0

- Michael Nacht, July 10, 2007
- Plaintiff's Expert Reports:
- James A. Strauchen, M.D., March 12, 2007 0 0 0
  - Brian G. Salisbury, M.D., March 11, 2007
    - Victor Roggli, M.D., July 27, 2007
- Victory Roggli, M.D., August 2, 2007

# Environmental Reports:

- o Mark F. Durham, CIH, June 1, 2007 o Kenneth A. Mundt, Ph.D., June 5, 2007

## • Medical Records:

- o Dr. Steven Greenstein
  o North Shore Pulmonary Associates
  o VA Medical Center Northport
  o Sloan Kettering Hospital

# Chronological Medical History

4/3/1923: Date of birth.

versus fibrotic changes at tight lung base. Subcentimeter nodularity in right upper lobe, may represent nodule or end-on vessel. Chest CT clarification is recommended. 5/19/2006: Nandita Wadhwa, Radiologist. Radiology Report. Impression: Focal atelectasis

٤,

6/23/2006; William O. Chu, Radiologist. Radiology Report. CI of the thorax. Abnormal chest x-ray. At the right base nodule lesions are identified contiguous with the lateral margin of the right hemidiaphragm; these nodules measure 1.7 cm and the largest measures 3.2 cm.

This cluster of nodule attaches to the right hemidiaphragm and likely represents sibronodular changes which may be associated with previous inflammatory disease. The remaining lung fields are clear without evidence of focal pulmonary parenchymal lesion. Hilar and mediastinal structures are within normal limits without evidence of adenopathy.

Below the diaphragm a right posterior renal cyst is present which measures 4.6 cm.

#### Impression:

Aggregate of nodules at the right base, contiguous with the pleural surface of the right hemidiaphragm

Etiology is uncertain but may represent a result of inflammatory process.

Cynthia Weiss Antonucci, Esq. August 22, 2007

7/20/2006: North Shore Pulmonary Associates. FVC – 3.04 (best), 87 (% pred); FEV1 – 2.32 (best), 86 (% pred).

7/31/2006: Jessica C. Lastig, M.D. Great Neck Radiologists. MRI Abdomen with and without contrast. Indication: Lung nodules in the region of the diaphragm, which are hypermetabolic on PEI/CI scan.

Findings: A cluster of nodular densities is seen at the right lung base along the right hemidiaphragm. There is no evidence of invasion into the liver. The liver appears homogeneous without focal abnormality.

#### Impression:

- Multiple nodular densities are again identified in the region of the right
  hemidiaphragm, as previously reported. Again, these are likely pleural based nodules.
  There is no evidence of hepatic involvement and no liver lesions seen.
- 2. Right renal cysts are identified.

8/16/2006; Valerie W. Rusch, M.D. The patient was seen by this primary physician and due to the patient's history of pleurisy one and a half years ago it was thought that possibly this was scartissue that attributed to that. A CT scan was obtained on 6/23/06 which revealed nodules at the right base measuring 1.7-3.2 cm. The patient was then seen by his primary pulmonologist, Dr. Denis Janus, who ordered a PET/CT on 7/25/06 which showed uptake in multiple peripheral nodular areas in the right hemithorax which were pleural based with an SUV of 3.1-5.7. There was a questionable area of hepatic involvement. Therefore an MRI of the abdomen with and without contrast was performed 7/31/06 which revealed multiple nodular densities in the right hemidiaphragm and right renal cysts but no areas in the liver. The patient is referred here by Dr. Janus for a possible surgical biopsy.

The patient smoked 2 packs per day for 18 years and quit smoking 47 years ago. He denies other tobacco use. He does not drink. He has known exposure to asbestos working in the floor covering business selling vinyl asbestos tiles. He did not install it although he sold it and handled it.

8/31/2006: Valerie.W. Rusch, M.D. Right video-assisted thoracic surgery, biopsies of diaphragmatic pleura, right lower lobe and right middle lobe wedge resection.

8/31/2006; Memorial Hospital for Cancer & Allied Diseases Department of Pathology.

- Biopsy diaphragmatic plaque (right) (fs) (pjm)
  - Diaphragmatic plaque #2 right (fs) (pjm)
- Biopsy right lower lobe mass lung (fs) (pjm) Wedge resection right middle lobe lung (fs) (pjm) Wedge resection right lower lobe lung (pjm)

#### Diagnosis:

- Pleura, right, diaphragmatic plaque; biopsy: Atypical mesothelial proliferation, favor epithelial mesothelioma.
- Pleura, right, diaphragmatic plaque #2; biopsy: Malignant epithelial mesothelioma.
- 3. Lung, right lower lobe mass; biopsy; Malignant epithelial mesothelioma.
- Tumor invades visceral pleura and is present on the margin of resection. Definite lymphovascular space invasion is not identified The tumor focally invades underlying lung parenchyma Lung, right middle lobe; wedge resection: Malignant epithelial mesothelioma Multiple foci of turnor are present 4;
- Immunohistochemical stain for calretinin is positive, supporting the diagnosis. Definite lymphovascular space invasion is not identified The tumor invades underlying lung parenchyma Lung, right lower lobe; wedge resection: Malignant epithelial mesothelioma ç,

resections, for resection of his suspicious right pleural nodules. He is making a smooth recovery 9/5/2006: Valerie W. Rusch, M.D. Addressed to Denise Janus, M.D. Just a brief note to bring assisted thoracoscopic surgery, pleural biopsies, right lower lobe and right middle lobe wedges you up to date on mutual patient Jack Nacht. On August 31, 2006, I performed a right video from surgery and was discharged home doing well on September 3, 2006.

9/18/2006: Lee M. Krug, M.D. The patient is an 83-year old gentleman with newly diagnosed mesothelioma referred by Dr. Rusch for treatment options.

Presented with right chest pain. The pain subsequently resolved and has not returned. More recently the patient was undergoing a routine physical examination at the VA Hospital. At that time a chest x-ray was performed and showed an abnormality near the right hemidiaphragm. This led to a CT scan dated 6/23/06 that showed nodules at the right lung base. He was referred to a pulmonologist who ordered a PET/CT scan dated 7/25/06 that showed multiple pleural based nodules with a SUV ranging from 3.1 to 5.7. There was a question of liver involvement and so middles with a subdomen was performed on 7/31/06 that did not show any involvement of the

The patient saw Dr. Rusch on 8/16/06. We underwent a diagnostic right VATS procedure on 8/31/06. The pathology revealed malignant epithelioid mesothelioma. The patient was felt to be inoperable due to his advanced age and he is referred for consideration of chemotherapy.

Past medical history significant for malaria in 1945 when he was in the Air Force during World War II and his plane was shot down. The patient has a history of BPM and he denies ever having a biopsy but says that his PSA is normal. He also had a basal cell carcinoma of the ear.

The patient lives on Long Island. He smoked two packs a day for 18 years, quit 47 years ago. He worked for a floor covering company selling vinyl asbestos tiles. He did not install the tiles but had significant exposure to them as a salesperson.

4/25/2007: Lee M. Krug, M.D. The patient had a CT scan of the chest performed on 4/16/07 in Florida. I did my best to compare it with his prior CT scan performed here. By my review, I feel there has been some slight decrease in the size of the pleural-based nodules, in particular the larger ones at the right base.

Impression/plan: The patient has completed two cycles of pemetrexed and carboplatin that he tolerated poorly, with multiple hospitalizations including after a 20% dose reduction. I explained the findings of the CT scan to the patient and we reviewed the situation. I suggested that we could continue the chemotherapy, but I offered treatment with just pemetrexed alone. The patient and his family are in agreement with this plan. Thus he will receive pemetrexed 400 mg/m2 every three weeks. The patient is already on the vitamin supplementation.

7/10/2007: Espeleta Vidal. CT scan. Ex-smoker 30 pack, years quit in 1959. No problems, pleuritic chest pain with deep inspiration which recurred 3 months ago, initially had it 2 years ago with diagnosis of pleurisy. Pain is slight, 1/10, dull pain. No fevers, chills, weight loss, hemoptysis, cough or chest pain. Never exposed to asbestos, never had pneumonia in the past flooring business sales only.

Past medical history includes hearing loss. He uses bilateral aides. Occupation: Flooring business, fighter pilot WWII, cruisers. Smoker - 2 ppd for 17 years; quit 1959.

7/27/2007: Victor L. Roggli, M.D. Duke University Health System. Surgical Pathology. The first specimen labeled S-06-33463, consisted of 41 glass slides and three paraffin blocks prepared from a pleural and lung biopsy specimen. These show an epithelial malignancy involving the visceral and parietal pleura. The tumor consists of sheets, tubules and nests in a desmoplastic stroma. Individual tumor cells are polygonal with anaplastic nuclei, prominent nucleoil, and moderate eosinophilic cytoplasm. Sections of lung parenchyma uninvolved by tumor show changes of centrilobular emphysaema, focal constrictive bronchiolitis obliterans, focal pulmonary ossifications and focal nuccous plugging with bronchiolar metaplasia. A few cosinophils are identified. The tumor cells stain moderately positive (2+) for cytokeratins using monoclonal antibodies AEI/AE3. The tumor cells stain moderately positive for calcutinn in both a nuclear and cytoplasmic distribution. Positive controls stain appropriately. Some sections show layers of a cellular hyalinized collagen arranged in a "basket weave" pattern, typical for parietal pleura plaque formation.

The second specimen, labeled KS-7078, consisted of six glass slides prepared from the same material as the first specimen. The tumor cells stain moderately to strongly positive (2+ to 3+) for calretinin in both a nuclear and cytoplasmic distribution. The tumor cells stain negative for LeuMI and carcinoembryonic antigen (CEA). Positive controls stain appropriately.

Additional sections were prepared from one of the paraffin blocks of tumor for further immunohistochemical studies using the avidin biotinylated complex technique on enzyme digested sections. The tumor cells stain strongly positive (3+) for cytokeratins 5/6, strongly positive for D2-40 in a membrane distribution and strongly positive for WT-1 in a nuclear distribution. The tumor cells stain negative for TTF-1. No appreciable staining is observed when an irrelevant mouse monoclonal antibody is substituted for immune serum. Positive controls stain appropriately.

By light microscopy, there were 37 asbestos bodies per gram of wet lung tissue (corrected for paraffin block). (2) This value exceeds our normal range of 0-20 AB/gm, and is within the range of values we have observed for patients with malignant mesothelioma and a history of occupational exposure to asbestos. (2) By SEM, there were 690 AB/gm and an additional 142,000 uncoated fibers per gram of wet lung (1000 x magnification). Twenty consecutive uncoated fibers were examined by BDXA, one of which consisted of magnesium and silicon in a proportion indicative of chrysotile asbestos. The remaining 19 fibers were not asbestos, and included 18 tale and one chromium. In addition, a single crocidolite fiber with a composition of Si-Fe-Na-Mg was identified among 205 fibers counted. A single asbestos body core was examined by EDXA, and this also was found to be crocidolite. The diameter of these two fibers was approximately 0.2 micron.

The gross distribution of tumor in this case as determined by chest roentgenograms, computed tomography of the thorax, and direct observations of the surgeon at time of thoracoscopy, when combined with the histologic and immunohistochemical feature so the tumor as described above, is diagnostic for malignant (diffuse) pleural mesothelioma, epithelial variant. (3) The additional findings of histologically confirmed pleural plaque formation and an elevated tissue asbestos content indicate that Mr. Nacht's mesothelioma is, in my opinion, related to prior asbestos exposure.

8/2/2007: Victor L. Roggli, M.D. Duke University Medical Center. It is my understanding that Mr. Nacht served in the US Army from 1941 to 1944 as a bomber pilot. From the end of 1945 until 1997, he owned a flooring company. He alleges exposures from breaking floor tiles to show customers. He also alleges exposure from being present while work sites were being cleaned up after tile installation.

Assuming this information to be true, and further assuming that he was exposed to any Calidria chrysotile, then it is my opinion to a reasonable degree of medical certainty that Calidria asbestos did not contribute to Mr. Nacht's mesothelioma. The reasons for this include the nature of Calidria chrysotile and the results of studies of experimental animals exposed to this form of asbestos. Calidria chrysotile is an unusual geologic formation of serpentine that is not contaminated by tremolite in the regions that were mined. The fibers in aqueous media tend to dissaggregate into small fibrils, nearly all of which are less than 5 um ilength. Aerosolized Calidria chrysotile fibers have the unusual property of increasing fiber diameter with increasing length. Thus only a very small percentage of fibers that exceed 5 um in length have diameters small enough to be respirable. In contrast with Casa that exceed 5 un in length have diameters surveys of the miners and millers or the Calidria deposit have not demonstrated the occurrence of asbestos related disease. Although the identification of numerous talo fibers is consistent with exposure to door tiles, the finding in this case of long thin crocidolite and chrysotile fibers suggest an exposure to asbestos cement products, such as cement pipe.

### Expert Reports

3/11/2007: Brian G.Salisbury, M.D., P.A., F.A.C.P., C.C.P. This 83-year old Caucasian male had an episode of pleuritic chest pain approximately two years ago, presenting with right sided chest pain. Apparently, from the records, the pain resolved and had not returned. A that time, a chest x-ray was performed, apparently in June of 2006, showing an abnormality near the right hemidiaphragm.

CT scan of the lung, dated June  $23^{rd}$  did show multiple nodules at the right lung base. This was pursued further. The patient had been referred to a Pulmonologist then ordered a PET CT scan dated July  $25^{th}$ , which showed multiple pieural based nodules with an SUV ranging

from 3.1 to 5.7. Although there was a question of liver involvement, MRI of the abdomen on July 31<sup>st</sup> did not show any involvement of the liver. On August 31<sup>st</sup>, the patient underwent diagnostic right VATS procedure. The pathology showed malignant mesothiclioma. The actual pathology showed that he had extensive pleural plaques and evidence of pleural mesothelioma involving the visceral and parietal pleura. The diaphragmatic pleura with a large multi-loculated mass adherent to the right lower lobe. Multiple biopsics are compatible with malignant mesothelioms with as bestos related pleural changes.

It is noteworthy that his FVC was normal at 89% of predicted. There was no evidence of any significant restrictive pulmonary defect.

In essence, this gentleman has extensive asbestos related pleural disease with mesothelioma. He did work for a floor covering company selling vinyl asbestos tiles and other asbestos containing flooring máterials. He did not install the tiles, but certainly had significant exposure to asbestos as a sales person.

With a reasonable degree of medical certainty, it is my judgment that this gentleman has asbestos related pleural malignancy.

3/12/2007; James A. Stauchen, M.D. Professor of Pathology and Vice Chair Director Anatomic Pathology. Mount Sinai School of Medicine. I have reviewed material on Jack Nacht, consisting of medical records, deposition transcripts dated November 21, 2006 and December 6, 2006, pathology reports and pathology slides.

Asbestos exposure/occupational history; Mr. Nacht was exposed to asbestos-containing floor tiles in the course of operating his carpeting and floor tile business from 1946 to 1997. He worked for a floor covering company selling vinyl asbestos tile. He did not install the tiles but had significant exposure to them as a sales person.

Radiology reports: Cheşt, 2 views PA and LAT, 5/18/06: There is hazy density at the right lung base adjacent to the costophrenic angel which may represent mild atelectasis or fibrotic changes. Impression: focal atelectasis versus fibrotic changes at right lung base. CT chest with contrast, 6/22/06: Impression: 1, Aggregate of nodules at the right base, contiguous with the pleural surface of the right hemidiaphragm. Bitology is uncertain but may represent a result of inflammatory process. Note: Further evaluation suggested and the consideration for biopsy recommended.

Pathology reports: Memorial Hospital for Cancer and Allied Diseases S06-33463: 1.
Pleura, right disphragmatic plaque; biopsy: Atypical mesothelial proliferation, favor epithelial
mesothelioma. 2. Pleura, right disphragmatic plaque #2 biopsy: Malignant epithelial
mesothelioma. 3. Lung, right lower lobe mass; biopsy: Malignant epithelial mesothelioma. 4.

Lung, right middle lobe; wedge resection: Malignant epithelial mesothelioma. Multiple foci of humor are present. The tumor focally invades underlying lung parenchyma. Definite lymphovascular space invasion is not identified. Tumor invades visceral pleura and is present on the margin of resection. 5. Lung, right lower lobe; wedge resection: Malignant epithelial mesothelioma. The tumor invades underlying parenchyma. Definite lymphovascular invasion is not identified. Imunohistochemical stain for calretinn is positive, supporting the diagnosis.

Pathology material: Memorial Hospital for Cancer and Allied Diseases: 20 H&E-stained slides S06-33463 1 (1), 2 (1), 3 (2), 4 (11), 5 (5). The slides labeled Memorial Hospital for Cancer and Allied Diseases S06-33463 1, 2, 3, 4, 5 are identified as "blopsy diaphragmatic pleural plaque", "diaphragmatic pleural plaque #2", "biopsy right lower lobe lung mass", "wedge resection right middle lobe", and "wedge resection right lower lobe". These show an epitheliloid malignant mesothelloma of the pleura invading the lung and diaphragmatic hyaline pleural plaque indicative of occupational asbestos exposure. Uninvolved lung shows patchy interstitial fibrosis. Ferruginous (asbestos) bodies are not identified, however, material for iron stains was not available. Report of immunohistochemical stains performed at Memorial Hospital for Cancer and Allied Diseases indicates the tumor cells are positive for calretinin. These findings are those of malignant mesothelioma.

Summary and conclusions: Mr. Nacht has a malignant mesothelioma of the pleura. Mr. Nacht had substantial asbestos exposures as indicated by the presence of diaphragmatic hyaline pleural plaque. Based on the recognized association of occupational asbestos exposure with the development of malignant mesothelioma and Mr. Nacht's history of occupational asbestos exposure as indicated in his deposition testimony and medical record, it is my opinion to a reasonable degree of medical certainty that occupational asbestos exposure was the cause of Mr. Nacht's malignant mesothelioma.

### **Environmental**

6/1/2007: Mark F. Durham, CIH. Golden Corporation, Occupational Health, Safety & Environmental Consultants. Mr. Nacht started Dee Jay Carpet as a carpet cleaning business in late 1945 with his father. Within months, the primary business of Dee Jay Carpet became the selling and installation of earpet and other floor coverings, primarily tile. The client base was both residential and commercial. Mr. Nacht remained with the business until his retirement 51 years later in 1997.

Mr. Nacht described the business as being originally 60 percent carpet sales and 40 percent tile sales. The tile sales were primarily to commercial clients, such as offices, commercial properties, and supermarkets. With the development of vinyl sheet flooring, some of the tile sales gave way to sheet flooring.

When asked what his responsibilities were during his 51 years in the business, he replied "Administrative and sales. I was a very, very good salesman." He stated further that "I did not install anything personally. I would supervise."

As a salesman of tile, linoleum, and sheet flooring, he described how sometimes be would show clientele the benefits of the products. For example, he would cut a piece of a sheet flooring from a cell to show how the color did not run all the way through the sheet. As the person in charge of Dee Jay Carpet, Mr. Nacht stated also that "he usually did the cutting" when a piece of sheet flooring was needed for an installation job. It was while cutting Mannington Mills sheet flooring that Mr. Nacht claims he could have been exposed to airborne asbestos.

On several occasions Mr. Nach was asked if there would be any dust generated when he cut the sheet flooring and his response, typically, was along the lines of "the dust would be so fine, you wouldn't see it" or "there had to be some." In other words, he did not testify that he saw dust released from the flooring as he cut it.

At odds with Mr. Nacht's testimony regarding his recollection of selling Mannington sheet floor is a statement made by Daniel Pranzo in his affidavit. Mr. Pranzo, an employee of Dee Jay Carpets for over 20 years starting in 1969, stated that he did not recall ever seeing or installing any sheet flooring manufactured by Mannington. It was also Mr. Pranzo's testimony that Mr. Nacht would only cut sheet flooring for installatioh jobs when there were no floor mechanics available to do the cutting.

Mr. Nacht's recollection of the absence of dust generated by outting sheet flooring is consistent with my experience. Industrial hygicne air monitoring data indicate that airborne filter levels measured during the cutting of sheet flooring are typically an order of magnitude below 0.1 floc. Estimating an 8-hour TWA exposure level for Mr. Nacht on a day during which he out sheet flooring is possible, but the resulting value may have little meaning because of the very small numbers involved. For example, assuming the cuts he made would have taken up to 60 seconds total, his daily 8-hour TWA exposure level would have been approximately 0.0002 floc.

\*

Mr. Nacht testified that in 1950 he bought and moved Dee Jay Carpet to a building located at 334 Central Avenue in Lawrence, New York. The business remained at this site through his retirement in 1997. Real estate records indicate that this building had a steam heating system that relied on an oil-fred boiler in the basement to generate the steam. Although the original date of construction for this building is not available, it was built certainly at a time when asbestos-containing insulation was applied to boilers and the steam-heat piping systems.

Mr. Nacht testified that he would go into the basement every week – presumably during heating season – to check on the boiler. When asked about the condition of the basement, he said that "It was a basket case. It was terrible. Everything was falling apart." Further, "It was dusty.

Very dusty. Incredibly dusty," Finally, when asked if the pipes were covered with pipe covering, he replied "Yes. In tatters."

The scene he described was a boiler room in a badly detentorated condition, with piping insulation that had been so damaged or so compromised over time that it was capable of releasing asbestos into the air of the boiler room. In fact, Mr. Nacht responded "Had to be" when asked if he was exposed to asbestos dust from the pipe covering. More importantly, when asked if he breathed that dust, his response was "I would have to."

The American Conference of Governmental Industrial Hygienists, in its documentation for the current Threshold Limit Value for asbestos, states "There is sufficient evidence to show that for a given level of exposure, the risk of developing mesothelioms is far greater with .....amosite than with chrysotile." During his weekly visits to the basement, it is very possible that Mr. Nacht was exposed to a mixture of asbestos fibers that could have included amosite as well as chrysotile asbestos.

In summary, it is my opinion that to a reasonable degree of scientific certainty the mixed amosite and chrysotile levels that Mr. Nacht could have experienced while checking on the boiler in the basement of 334 Central Avenue could have been several orders of magnitude greater than any exposure level he could have experienced while cutting Mannington sheet flooring. Further, the contribution to his lifetime asbestos exposure resulting from the weekly basement exposures over a period of 47 years would be far greater than any contribution that could have come from his occasional cutting of Mannington Mills sheet flooring.

6/5/2007: Kenneth A. Mundt, Ph.D. Principal and Director of Epidemiology ENVIRON International Corporation. Based on my review, analysis and synthesis of the published epidemiological, occupational health and case-specific information available to me, and assuming that Mr. Nacht actually sold Manninngton Mills stock containing chrysotile asbestos. I concluded to a reasonable degree of epidemiological certainty that Mr. Nacht's alleged handling and cutting of rolled vinyl flooring produced by Mannington Mills did not cause Mr. Nacht's mesothelioma. While the literature indicates that substantial numbers of idiopathic pleural mesotheliomas occur, regardless of prior exposure, Mr. Nacht's pleural plaques suggest that he sustained some past exposure to asbestos, possibly amphibole asbestos. The sporadic potential exposure to low-level chrysotile from handling and cutting Mannington Mills sheet goods containing chrysotile asbestos would have been negligible and of no consequence with respect to his pleural mesothelioma.

# Interrogatories - 10/6/2006

Jack Nacht is 83 years old. His date of birth is April 3, 1923. He is a widower. His father's name was David Nacht and he passed away in 1963, at the age of 70. The cause of death

was kidney cancer. His mother's name was Bella Nacht and she passed away in 1979, at the age of 86. The cause of death was a stroke.

From 1997 to present, Mr. Nacht has resided in a single family unit in Boca Raton, Fl. Electric is used for heating and cooking. He has not had any significant home improvements done.

From 1987 to 1997, he resided in a single family unit on 554 Fairway Drive in Woodmere, NY. Electric was used for heating and cooking. He did not have any significant home improvements done.

From 1960 to 1986, he resided in a single family unit at 28 Woodmere Blvd. South in Woodmere, NY. Oil was used for heating. He had bathroom and kitchen renovations done.

From 1953 to 1960, he resided in a single family unit in Wantagh, NY. Oil was used for heating. The den was renovated.

From 1923 to 1953, he resided in various residences in Queens, NY. Electric was used for heating and cooking. No renovations were done.

Mr. Nacht was diagnosed with mesothelioma in August of 2006. At various and numerous times, he has experienced a variety of different and differing symptoms related to his injury which are numerous and frequent.

٠.

Since his diagnosis with mesothelioma, Mr. Nacht has experienced increasing shortness of breath, mental and emotional distress, and inconvenience. Mr. Nacht's asbestos-related condition has disrupted his life, limiting him in his everyday activities and interfered with his living a normal life.

Mr. Nacht has had biopsies and/or tissue samples taken at Memorial Sloan Kettering Hospital, but the medical records may or may not reflect other locations.

Mr. Nacht smoked cigarettes between 1942 to 1960. He smoked one pack per day. He was never told by a physician that he is or was suffering from any disease or illness caused by or contributed to by tobacco. He was never advised by any physician that use of tobacco products could adversely affect his health and that he should stop using tobacco products. His wife smoked cigarettes from 1945 to 1960s.

Mr. Nacht was enlisted in the Army Air Force from 1942 to 1945.

He recalls having been exposed to a variety of different asbestos containing products while self employed, including but not limited to: pipecovering, block, cement, cloth/clothing, gaskets, firebrick, tape, sheetrock, compound, floor tile, celling tile, and insulation used on equipment; which were manufactured by various companies, some of which he may identify at his deposition.

He voluntarily retired in 1997.

He states that he was never warned of the harmful effects of exposure to asbestos.

#### Depositions

# Jack Nacht taken on November 21, 2006

Mr. Nacht was born on April 3, 1923. He is 83-years old. For the past three years he has lived in a condo at 139 Darters Lane in Manhasset. He does not own the condo.

Mr. Nacht was married to Rita Horowitz in 1947. Rita had lung cancer and passed away in 1996. That was his only marriage. Rita was a smoker in 1947. She smoked un-filtered Camels. They both smoked in the family home. Rita stopped smoking in 1959.

Mr. Nacht started smoking at the age of 19 and stopped at the age of 36. He smoked a pack and a half of un-filtered Camels a day. He states that he stopped smoking because he got irritated when he realized that he could not control it.

Mrs. Nacht was a school teacher while they were married.

Mr. Nacht's son Lawrence, suffered from thyroid cancer at the age of 12, but was successfully treated. His father had kidney cancer.

Mr. Nacht enlisted in the United States Army in 1941. He went to a small town in Georgia and was designated to be an aviation cadet. He went to school for two months and received training how to fly a plane. He never learned how to perform mechanical repairs.

He lived at 28 Woodmere Boulevard South for 30 years. He moved in about 1960 and remained there until 1990. He hired a contractor to renovate the bathrooms when he first moved in. He does not recall the name of the contracting company. He is not certain as to whether or not he was exposed to asbestos while living in that home.

In 1990, he moved to Fairway Drive in Woodmere and remained there for approximately six to eight years.

Cynthia Weiss Antonucci, Esq. August 22, 2007

Mr. Nacht started Dee Isy Carpet Company in 1945. He started the business as a rug cleaning business. He personally cleaned wall-to-wall carpets in commercial and residential buildings. In 1946 he decided that he was going to sell carpet. He figured it would be more profitable. After the first eight or nine months, he started to sell carpet and tile. He personally sold carpet and tile, and measured floors. He worked in the store. 50% of the time he went out and measured for the carpet installation. At times he sent sales people to measure and install the carpet and tile. His sons worked for him. Mr. Nacht never installed carpet. 60% of the business was installation of carpets and 40% was installation of tile. He sold carpet and tile at Dee Isy Carpet until he retired in 1997.

Dee Jay Carpet started selling tile in the late 1940s. In 1947, 15% of the business was tile sales. He never installed any tile. He ordered tile from Amtico, Armstrong, Azrock, Kentile, Matico, Johns-Manville, Goodrich and Mannington. In 1948 he sold a smaller percentage of tile than later. In those days Kentile was the only manufacturer of tile. In 1948 he opened up another store in Lynbrook where he sold tile, linoleum and carpet.

At some point during the 1960s, he stopped selling asphalt tile. He wanted to concentrate on better merchandise. Vinyl asbestos was a much better product. It looked better and was much more expensive and it sold better. Kentile made asphalt tile and vinyl asbestos. He does not recall if he personally ordered the asphalt tile from Kentile or if his sons did it.

The first time Dee Jay Carpet sold Kentile of any kind was in 1948 or 1949. He does not recall the last time Dee Jay Carpet sold Kentile. His employees would order the Kentile asphalt tile from Eastern Tile. He believes that the Kentile asphalt tile probably contained asbestos. Other than his son Michael, telling him about a month ago that the Kentile asphalt tile could have contained asbestos, he does not have any other reason to believe that that thie contained asbestos.

He stated that the primary focus when he would go out to residences to inspect the tile work was to make sure that the aesthetic tile itself looked good. At least 50% of the job would be finished by the time Mr. Nacht would go to inspect it. Commercial settings were simple as a rule. The only problem he had was making sure it was done right and that any design was correct. He also had to make sure that the workmen were going to clean up the job. He stated that the cleanup was important because he wanted to make a good impression. He always got paid after the job was 99% finished. When he visited a residential job site at which his tiles were being installed, he would stay until they had about an hour to go. The most amount of time he ever spent at any commercial site was an hour. He usually went at the end of the day when 90% of the work had been completed.

He found out the when he would break a tile in the store to show a customer the resiliency of the tile, that a certain amount of asbestos would come off the tile where he broke it. He is not certain how many time he broke tiles, but it could have been a hundred times. During

his 45 years in the business, he sold tile every day. Not every customer wanted to see the tile broken and not every tile could be broken. He stated that the vinyl tile and asphalt tile could not be broken. The vinyl asbestos tile was the only tile he could break. The only way he was exposed to asbestos while in his store was when he was breaking tile. He broke it in half in approximately a second. After it was broken in half he would show the customer how the colors ran through: After he had shown it to the customer he would throw it away. The entire process would take a minute or a minute and a half.

He believes that he was exposed to asbestos when he went into the field because he would go on the jobs and all the curting was done along the walls and there would be a certain amount of dust collecting on the floor. He would make sure the place was cleaned up. He would stand there and tell the workers to clean up and they would start cleaning while he was still standing there. The dust would rise up and that is when he believes he was exposed to asbestos.

# Jack Nacht taken on December 4, 2006

He stated that when cutting tile, being that it came in small pieces, there were more cuts than with big pieces. When linoleum was used, the clean up job consisted of taking a broom and a dust pan and sweeping it up. It was always dry sweeping.

 $\ensuremath{\mathrm{Mr}}$  . Nacht and his employees never used any respiratory protection because they never knew there was a problem.

ķ.

On an average day, Mr. Nacht would come in the morning, check to see that the mechanics were there to do the work, make sure the materials were available, make sure they were loaded on to the trucks, give out floor plans, give directions to the jobs, give instructions as to special caré and let the mechanics know that he would see them later in the day. If the job took half a day to do, he would go and inspect it in time just before the men finished. If the job took a full day to complete, he would go there towards the end of the day. He employed 12 mechanics. He would go to more than one work site per day. Sometimes he went to three or four worksites in a day. The worksites were mostly residential.

As a rule, Mr. Nacht usually asked for outside contractors to be cleared out before his mechanics came in to install the carpet and tile. He would never be at a worksite with sheet rockers or pipe coverers.

Mr. Nacht would go out and inspect every commercial job whether it was carpet or tile. He is not able to recall if the clean up would start after the cove base was down.

His residential jobs consisted of 50% carpets and 50% resilient floors. When he had an installation at a residential site, he did not visit the site every single time.

Barlier in his deposition he stated that approximately 60% of his business was carpet and 40% was resilient floors. That percentage is based on later years.

Dee Jay Carpet began selling vinyl asbestos tiles as soon as they became available in the early 1950s. In the beginning it was just Kentile, which was asphalt tile and then it got better with the vinyl asbestos.

Mr. Nacht believes that he was exposed to dust whenever vinyl asbestos tiles and asphalt tiles were installed in his presence. His mechanics would use a dust pan, a broom and sometimes a towel to clean up. Mr. Nacht believes that he came in contact with the dust and breathed the dust. He was also exposed to dust released from vinyl asbestos tile whenever he needed to show a customer the composition of that tile. He had to break the tile to show the customer that the color ran through from top to bottom. That process would cause dust to be released. He never saw the dust but it was there. Dust was also released from Mannington Mills linoleum sheet and linoleum whenever he cut that. He would be three inches from the cut and he did the cutting many times.

In 1950, Mr. Nacht opened a second store. That store was heated with oil heat and the boiler was located in the basement. There were pipes running from the boiler. He had to go down to the basement every week to check on the boiler. The basement was falling apart and it was very dusty. The pipes were covered with pipe coverings in tatters. He believes he was exposed to the dust from the pipe covering.

# Michael Nacht taken on July 10, 2007

45

When Mr. Nacht was out on a job with the mechanics, they would teach him how to work. He did some hands on things with their supervision. He installed carpet and tile. He did everything that could possibly be done.

After he became a full time employee of Dee Jay Carpet, there was no one else the mechanics would bring out to the jobs to clean up. Whoever was doing the job, would do the clean up.

Between 1970 and 1971, Dee Jay Carpets sold carpet, linoleum and tile. From 1972 until his father retired, Dee Jay Carpets sold and installed linoleum, carpet and tile. They sold tiles including viryl, viryl composition tile (VCT) and viryl asbestos tile (VAT). He does not recall what year regulations stopped the maurfacturers from making VAT. The manufacturers that made the viryl asbestos tile included Kentile, Amtico, Azrock, Johns-Manville and Mannington. Dee Jay Carpet stopped selling asbestos tiles when the government would not allow anyone to manufacture them anymore.

Cynthia Weiss Antonucci, Esq. August 22, 2007

He recalls his father's responsibilities to include taking care of the bills and selling. His father would also go out and check on the jobs and the men. Mr. Nacht would stay in the store. As the time progressed he recalls his father spending more time out of the store than in it. He went to check on the men daily. Through junior high school and high school Mr. Nacht spent time going out with the mechanics to the sites and he would see his father out at these sites. At times, his father would go in and come out very quickly from a site and other times, he spent two hours at a site depending on the job and what was going on. While out on the sites, his father would tell everyone what to do and overseeing the jobs. He never installed carpet or tile. He paid åttention to the clean up. Mr. Nacht stated that the biggest problem in his industry is getting mechanics to clean up behind themselves. Mr. Nacht sold more carpet than tile.

When he was doing work on job sites before college, on every 20 jobs, there were other trades including electricians, carpenters and painters, working around him and also when his father would be visiting. He does not know if any of the materials the other trades used contained asbestos. He does not recall ever having seen anyone working on pipes when he was at these jobs. He never saw any workers using pipe insulation. He has never seen pipe insulation on a job site.

He does not recall ever having seen any warnings or having gotten any materials from vendors or manufacturers of resilient flooring. He did not get any installation guides or how-tos from the manufacturers. He never belonged to any of the flooring organizations like the Resilient Floor Covering Institute.

### CONCLUSIONS

4

# Presence and Extent of Alleged Injury/Illness

Mr. Nacht was diagnosed with malignant epithelial mesothelioma in August 2006 at the Memorial Hospital for Cancer and Allied Diseases in New York (Sloan Kettering). This diagnosis was confirmed by pathologist, Victor Roggli. His medical history includes basal cell carcinoma and pleurisy 1.5 years prior to his diagnosis of mesothelioma.

Evaluation of pulmonary tissue by Dr. Roggli showed both free and coated ("asbestos body") crocidolite fiber and confirmation of the presence of pleural plaques.

#### Exposure

Mr. Nacht testified that he believes he was exposed to asbestos through floor tile selling and his presence at installation of floor tiling and especially the sweeping/clean up of the dust generated at these jobs, the majority of which were residential. He further testified that he worked 60 percent of his time with carpeting and 40 percent with floor such as tiles. Mr. Nacht

stated that he would sell floor tile everyday and that he would often break the tiles in half to show the customers that the color ran through from top to bottom; however this took one to one and one-half minutes and then he would throw away the broken tile. His exposure to alleged asbestos-containing dust occurred when he would inspect residential and commercial floor tile jobs to make sure that the job was performed properly along with the clean up/sweeping. He would frequently go to the job and would stay for an undetermined amount of time, except that he specified he would remain at a commercial site for less than one hour.

In his answers to interrogatories he recalls having been exposed to a variety of different asbestos containing products while self employed, including but not limited to: pipe-covering, block, cement, cloth/clothing, gaskets, firebrick, tape, sheetrock, compound, floor tile, ceiling tile, and insulation used on equipment. Mr. Nacht did recall at deposition that he would check the boiler in his store basement weekly and that the basement was very dusty and further that the insulation around pipes in the basement were in tatters. Specific exposure to block, insulation used on equipment, cement and other sources of potential amphibole exposure has not been provided in the information provided for review and evaluation.

## Causal Association

It is my opinion that Mr. Nacht's unfortunate mesothelioma was not the result of work with or in and around asbestos-containing floor materials. It is my further opinion that his tumor if indeed the tumor is asbestos related at all, is the result of exposure to amphiboles from other non-flooring related, asbestos exposures, likely amphiboles from the work around boiler insulation, other amphibole substantial exclusion becontaining products. Insulation in the United States contained substantial levels of the amphibole amosite, and amosite is the most commonly found fiber type in the lungs of those with mesothelioma; however, substantial amounts of crocidolite have also been imported into the United States and however, substantial amounts of crocidolite have also been imported into the United States and Nacht and found it to be crocidolite. The evidence from the scientific literature reflects that the vast preponderance of asbestos bodies have amphiboles as the fiber type comprising the asbestos body core (Murai et al., 1995; Toassavainen et al., 1994; Warnock and Wolery, 1987; Roggli and Sanders, 2000). Thus, in conjunction with the free crocidolite fiber in the limited sampling of Mr. Nacht's lungs, the asbestos body containing the crocidolite is strong evidence for an unreported and heretofore likely etiology for Mr. Nacht's mesothelioma.

Over twenty studies (see references) of removal of existing asbestos-containing floor covering using recommended procedures as well as installation and use of such floor covering have shown from the 1970's through the 2000's that exposures to floor tile work has not been found to produce exposures in the range where increased risk of asbestos-related disease has been identified including for mesothelioma. Generally their eight (8) hour, time weighted average

exposures are in the range similar to that of brake/auto mechanics, at 0.02 – 0.002 f/co. Further, it is noted that Mr. Nacht said that he only inspected and supervised his employees on site for a small part of the work day and otherwise would only briefly break tiles in his store to show customers the color feature. This would severely limit the fiber year cumulative dose that Mr. Nacht would have received from that particular potential asbestos exposure source. I have no data or appropriately derived scientific methodology indicating that Mr. Nacht's experience in and around asbestos-containing tiles would produce an increased risk of developing any asbestos-related disease including mesothelioma, or pleural findings/e.g., pleural plaques). The presence of pleural plaques indicates a significant, asbestos exposure. It would take an exposure and likely an amphibole exposure not consistent with that possible from working full-time with floor tile much less the limited exposure potential described by Mr. Nacht. Evidence from the scientific literature confirms the increased risk of pleural plaquing from amphibole as compared to chrysotile asbestos exposure (churg and DePaoli, 1988; Churg et al, 1993; Boutin et al, 1989; Manda-Stachouli et al, 2004; Churg, 1982; Wannock et al, 1922).

As knowledge of the potential for exposure to airborne asbestos fibers from work with asbestos-containing materials such as braking material and vinyl asbestos tile began to be published in the 1970's and concern for health risk at lower levels of exposure than previously thought grew during that same time frame and beyond, various studies have been performed which shed considerable light on the level of risk for the development of mesothelioma among workers performing these activities. Information concerning mesothelioma risk comes from three scientific pursuits:

 Epidemiological studies of workers performing brake repair and replacement and other activities, e.g., grinding and drilling.

1

- Lung tissue fiber burden analysis of various "exposed" and "unexposed" workers and general population individuals with mesothelioma.
- Formal risk assessments using epidemiological studies to predict risk of developing mesothelioma at low levels of exposure.

Key factors surrounding issues of potential for mesothelioma development secondary to work with asbestos-containing floor covering center around the following:

- Chrysottle asbestos was used virtually exclusively in the production of floor covering materials.
- Epidemiologic studies of "pure" chrysotile study populations (cohorts) do not show elevated rates of mesothelioma for such populations.

- mesothelioma in so-called "pure" chrysotile-exposed populations is likely related to exposure to the contaminant amphibole fiber, tremolite, in those operations. Scientific evidence from the study of chrysotile miners/millers indicate that risk of
- Although not totally removed from the milled chrysotile product used to manufacture similar workplace exposures to floor mechanics shows similar levels of tremolite in the asbestos-containing floor covering, fiber burden analysis of brake workers with removal was sufficient to avoid appreciable exposure to tremolite fibers to those workers as compared to members of the general population indicating that the working with chrysotile-containing products. •
- Studies of fiber type/burden in patients with mesothelioms virtually always show the presence of amphibole fibers, primarily amosite, tremolite and crocidolite.
- Actual epidemiological studies of brake workers who again have similar asbestos exposures to floor mechanics do not show an increased risk for the development of mesothelioma.
- Aithough the exact level of what can be termed "non-asbestos related mesothelioma" is debated, at least ten to twenty percent of mesotheliomas are not the product of

work. The relative risk or odds ratios and corresponding 95% confidence intervals for each study Seven epidemiológical studics, all case-control studies, provide no scientific evidence for an increased risk of mesothelioma in auto mechanics exposed to asbestos from brake and clutch along with the number of mesothelioma cases and dates of diagnosis are provided asbestos exposure. chronologically: McDonald and McDonald (1980) - 480 cases in Canada (1960 - 1972) and United States (1960) - RR/OR - 0.91 (0.39 - 2.13)

Spirtas et al (1985, 1994 update) – 259 cases in NY and LA (1975 – 1980) RR/OR – 1.00 (0.60 – 1.60)

Teta et al (1983) - 220 cases in Connecticut (1955 - 1977) - RR/OR - 0.65 (0.08 - 5.53)

Woitowitz and Rodelsperger (1994) – 324 cases in Germany with motor mechanics beginning employment 1925 – 1972 – RR/OR – 0.87 (0.46 – 1.64)

Teschke et al (1997) – 51 cases from British Columbia (9/90 – 8/92) · RR/OR – 0.80 (0.20 – 2.30)

Agudo et al (2000) - 132 cases in Spain (1993 - 1996) - RR/OR - 0.62 (0.17 - 2.25)

Hessel et al (2004) – expanded Spirtas studies of 1985 and 1994 – RR/OR – 0.71 (0.30 – 1.60)

A meta-analysis of these case-control studies by Otto Wong published in 2001 reported a relative risk for all studies combined of 0.90 (0.66 – 1.23). It should be noted that the relative risk value for the Agudo study was calculated by Dr. Wong. The Goodman et al. 2004 meta-analysis also noted no increased risk for mesothelioma. A recent article by Lemen in 2004 calls these findings into question; however, there are numerous deficiencies in his approach limiting the applicability of his opinions. Harvard researchers, Laden et al also in 2004 performed a critical review of all automobile mechanics epidemiological studies and stated "...evidence did not support an increase in risk of either lung cancer or mesothelioma among male automobile mechanics occupationally exposed to asbestos from brake repair." Finally, Butnor et al in 2003 reported on fiber lung burden in 10 cases of brake-dust exposed individuals. Although the exposure for the significant amphibole presence was not reported, a frequent problem in asbestos studies, all ten have excess commercial amphibole fibers in their lungs.

document are interesting, but they have not been reported in the open literature and it is unclear if Woitowitz and Rodelsperger, 1991; Langer and McCaughey, 1982; Huncharek et al, 1989) have aithough one cannot say that there is a deficit of risk as the confidence intervals does not provide nature and especially when compared against more formal epidemiological investigations that do statistical significance. It should be stressed that the time frames of exposure occurred primarily discussed above) has not shown an association. Case reports/series are hypothesis-generating in populations through North American and two European countries providing further evidence of consistency in different study populations and conditions. While earlier case reports or series that some cases of mesothelioma will occur simply due to chance. The three case reports by  $\mathrm{D} r$ not have the ability in and of themselves to demonstrate association or causation. It is expected other exposures were potentially operative, if the diagnosis was confirmed and other issues of when compressed air for "blowing" out brake dust was used. As noted previously, this would reported limited findings warranting further study, such study (i.e., case-control publications There is consistency among all of the studies that report a relative risk of one (1) and usually considerably less. Relative risk of one or less indicates absence of increased risk correspond to the potential for highest asbestos exposure. Additionally, the studies examine Grace Ziem referenced in Dr. Castleman's textbook and cited in the 1996 BPA guidance causation were satisfied.

Studies exploring fiber type and burden in individuals with mesothelioma have been performed by a number of investigators. These studies are useful in exploring the presence of amphibole fibers versus ohrysotile fibers. However, such studies while consistently demonstrating exposure to amphiboles, dose response data comparing amphibole burden versus ohrysotile burden cannot be confidently generated due to ability to eliminate chrysotile fibers

Cynthia Weiss Antonucci, Esq. August 22, 2007

preferentially over amphibole fibers as amphibole fibers are much more likely to be retained in lung tissue over the years. Nonetheless, such studies do call into question, those opinions which attempt to relate mesothelioma to chrysotile exposure especially at extremely low exposure levels such as that which has been demonstrated to occur in brake repair and replacement workers (see exposure section above). Beginning in the early 1980's research by McDonald in Canada and assessment by Churg in 1982 along with substantive studies by Churg, Roggii and others, yielded information regarding the fiber types and levels of fibers in the lungs of patients with mesothelioma. Such studies showed that amphiboles were virtually always present and in appreciable levels especially in comparison to chrysotile fibers. Churg in 1982 stated:

"Analysis of pulmonary fiber burden suggests that asbestos-related disease is not merely a matter of total number of fibers represent, but that factors such as fiber type and size are equally important."

Further commenting mesotheliomas, Churg stated:

"These observations suggest that most mesotheliomas are associated with numbers of increased numbers of commercial amphiboles and not with chrysotile asbestos."

Churg et al in 1984 performed a fiber analysis of lung asbestos content in five chrysotile (Quebec) workers with mesothelioma. This study of six (6) patients found both tremolite and chrysotile in the lung tissue however, the tremolite levels were 9.3 times the controls and concluded that tremolite exposure/content may be important in the pathogenesis of these tumors.

16

McDonald et al in 1989 reported on 78 Canadian mesotheliomas diagnosed 1980 – 1984 attributing approximately 68 percent of the Canadian tumors to amphibole fibers, especially tremolite, and virtually no mesotheliomas to chrysotile fibers. Churg and Wright also in 1989 evaluated nine matched pairs of mesothelioma patients who were either shipyard and insulation workers, or chrysotile-industry workers. The researchers report that a relatively lunge chrysotile content would be necessary to induce mesothelioma on the order of three times that necessary for asbestosis development. However, the amphibole level associated with mesothelioma was 1/35 asbestosis development. However, the amphibole level associated with mesotheliona was 1/35 ability of amphiboles and chrysotile to produce mesothelioma. Gibbs et al in 1990 reported on 10 paraoccupational cases of mesothelioma patients in England whose husbands had worked with asbestos. The fiber analysis showed only one person with increased chrysotile burden however that individual also had an increased level of amphibole fiber as well. Two individuals on 221 mesothelioma cases in Australia and found only two cases with "chrysotile only" fibers in the lung tissue with the authors concluding that the greatest risk for mesothelioma in Australia was exposure to crocidolite, especially those fibers greater than 10 um. Interestingly, there was only a significant dose response effect for short chrysotile fibers which is not consistent with

regulatory or risk assessment evaluations (see Risk Assessment below). Churg et al in 1993 reported on 19 chrysottile miners/millers with mesorheliomas noting that the mesorhelioma burden is probably tremolite-related and that the role of chrysottile remains uncertain. Sakai et al in 1994 reported on 16 mesorhelioma patients gathered over 1983 to 1990 from 15 Japanese hospitals. All 16 have significant pulmonary fiber levels of amosite and/or crocidolite.

A 1995 study by Dufresne of 12 Canadian chrysotile asbestos miners with malignant mesotheliomas. Two of the 12 cases were felt to be attributable to important crocidolite and amosite fiber, while the other ten where associated primarily tremolite or a tremolite/chrysotile combination. All twelve had tremolite fiber present. In 38 mesothelioma cases in Canadian asbestos miners, millers and factory workers from the Thetford and Asbestos operations, McDonald et al reported in 1997 that all had tremolite in the lung tissue except two; however, those had crocidolite. The authors noted that the predominance of the cases were from the Thetford mines and those workers had generally worked in specific areas where tremolite presence was considerable. Some workers had also been exposed to crocidolite and amosite fibers, apparently used in factories for production of gas masks. The enhanced retention of tremolite fibers from the lungs of workers in the Thetford mines was confirmed by Nayebzadeh et al in 2001.

٠,٠

٠,

Some studies have reported that mesotheliomas are present in individuals with chrysotile only in the lung tissue. Nolan et al in 1994, reported that in one case of mesothelioma with high levels of chrysotile in the lung tissue that amosite and crocidolite fibers were not found. The authors specifically conclude that they found no evidence of "commercial amphiboles." However, the authors report that almost one (1) percent of the fibers were silicate fibers also known as the contaminant tremolite. In 2002, Suzuki and Yuen reported on the fiber types and dimensions found in the lungs and mesothelioma tissue of 168 patients with mesothelioma. These patients had a variety of exposures to asbestos although specific associations with exposure history were not reported. While some patients had chrysotile only in their mesothelial tissue. It is critical to note that the researchers included very small fibers, i.e., shorter than one micron in length. Such fibers have not been implicated in the production of asbestos-related disease including mesothelioma. The mere presence of short chrysotile fiber in tumor tissue does not indicate causation, merely that fibers of that dimension can gain entry to tissues outside the lung. Additionally for the 11 patients with chrysotile only in their lung tissue, the fiber size is not identified. This study has received considerable criticism in the scientific arena. Thus, an association between fibers of the size considered to be pathogenic was not indicated for exposure to chrysottle fibers alone.

Roggli et al reported in 2002 on 1445 mesothelioma cases and fiber type and burden in 268 of those cases in correlation to their occupational and environmental exposure to asbestos. As in other studies of this study population type, the predominate fiber found was amosite. No cases were reported as having chrysotile alone. Importantly, the fiber levels of amphiboles,

Cynthia Weiss Antonucci, Esq. August 22, 2007

mesothelioma. It is important to note that in this study elevated lung levels of <u>commercial</u> (emphasis added) amphiboles in some brake workers suggest that unrecognized exposure to these exposures and therefore were not at increased risk over "unexposed" populations. The authors noted "brake dust is unlikely to cause mesothelioma." Dodson et al, in 2003 reported on asbestos Chrysotile was not reportedly found in 11 cases and there were no cases of chrysotile fiber found workers in the automotive as those whose only possible exposure to asbestos was having been building occupants. The eleven automotive brake repair workers in the automotive category had burden and fiber type in 15 women with mesothelioma who had worked in various occupations. alone. Butnor et al, in 2003 reported on fiber burden in 10 cases of brake repair workers with especially noncommercial amphiboles, and chrysotile found in lung tissue were similar for background," indicating that such workers who developed mesothelioma did not receive The most commonly found fiber was amosite and the next most common was tremolite. "either an elevated amosite content or an asbestos concentration indistinguishable from ibers plays a critical role in the development of MPM.

Retrospective cohort studies of workers considered exposed to only chrysotile asbestos chronologically showing the number of mesotheliomas and noting amphibole contamination: also provide evidence as to the inability of this fiber type to produce mesothelioma. Nine studies, purported to represent "chrysotile-only" exposed worker cohorts, are outlined

1977 Weiss

٠.

Robinson and Lemen 1979

264 workers

3276 workers

No mesotheliomas

17 mesotheliomas

Note: Exposure to both amosite (5 percent during three WWII years) and crocidolite (< 1 percent during the three WWII years). Other year's amosite constituted 1 percent. Note: The mesothelioma was not supported by histological examination and the 1 mesothelioma 952 workers, 1979 Rubino et al

calculated and measured.

exposures to what was termed "pure" chrysotile were reported as very high when both

Asbestos and Thetford. As noted previously in this report, chrysotile asbestos from these Note: The study population worked in the Canadian asbestos mines in the towns of mines had significant levels of the amphibole tremolite and tissue fiber burden/type analysis in the mesotheliomas from this population note the significant presence of 11 mesotheliomas 11,379 workers tremolite in those individuals. 1980 McDonald et al

between another similar workforce where that fiber was used in gas mask manufacturing. Acheson et al. 1982 570 workers I mesothelioma
Note: Exposure to crocidolite asbestos is possible as there was some interchange Acheson et al.

٠,

Cynthia Weiss Antonucci, Esq. August 22, 2007

Note: Authors noted that only an "insignificant" quantity of asbestos fiber other than 1 mesothelloma 768 workers Dement et al.

Note: Some amosite and a small amount of crocidolite were used in the Pennsylvania 14 mesotheliomas chrysotile was ever processed. Specific amounts were not mentioned 4137 workers plant where the exposures occurred. McDonald et al.

crocidolite exposure. One of the two remaining cases may have had "fringe" exposure to Crocidolite had been used in pipe manufacture and that worker's compensation case had Note: Bight of the ten individuals with mesothelioma had definite exposure to crocidolite and the other had previously worked at an asbestos cement factory. 10 mesotheliomas 11,182 workers been attributed to his earlier work. Newhouse et al.

Note: Crocidolite asbestos had been used at the asbestos cement factory prior to 1936 and both individuals with mesothelioma had been employed there during the use of 2 mesotheliomas 1970 workers 1982 Thomas et al. crocidolite.

previously, support this point as even in populations highly-exposed to chrysotile fibers, mesothelioma is produced in those with brief commercial amphibole exposure and work in areas especially short ones, in an extremely limited number of "chrysottle only" cases is insufficient to noncommercial amphiboles in mesothelioma cases, absence of mesotheliomas when discounting illustrated by "background" populations with numerous asbestos fibers of all types per gram of lung tissue (Dodson et al, 1991) and risk assessment studies showing no increased risk at such levels (see discussion below). It is clear that amphibole asbestos exposure can indeed produce mesothelioma at relatively brief and low levels of exposure. The mining/milling studies cited In contrast to the hypothesis put forth by Smith and Wright in 1996, where the authors performed cohort, case-control and fiber burden/type epidemiological studies especially those causally associate chrysottle exposure alone with the development of mesothelioma. This is involving considerable numbers of cases consistently show the presence of commercial and amphibole exposures and lack of increased risk of mesothellomas especially in populations exposed to low levels of virtually pure chrysottle. The mere presence of chrysottle fibers, speculate that most mesotheliomas are the result of chrysottle exposure, numerous wellwith tremolite fiber contamination. It is also important to note that of the roughly 57 mesotheliomas that were reported in the In two cohorts there were no mesotheliomas as the tumor in the Rubino et al 1979 study was not "chrysotile only" cohorts, there was evidence for amphibole exposure in virtually all instances. histopathologically confirmed. Another critical point is that while there was no pathological

the limited amphibole usage and expected exposures are consistent with the various assessments and reports of environmental and paraoccupational amphibole exposures as capable of producing mesothelioma at levels considerably lower when compared to occupational exposures. Yano et al, in 2001 reported a cohort study of 515 male asbestos workers who apparently worked with amphibole-free chrysotile and noted that heary exposure (4.5 and 7.6 f/cc) to pure chrysotile can possibly cause lung cancer and malignant mesothelioma. A meta-analysis of 26 mesothelioma deaths from various studies reported an increase of mesothelioma in chrysotile-only exposed workers; however, there is no mention of potential amphibole exposure as discussed above (Lu et al, 2004). However, Burdorf et al (2005) noted the difference in pleural mesothelioma rates between Sweden and The Netherlands and proposed that the difference was likely explainable due to proportion of exposed individuals, exposure level differences and type of asbestos fiber.

Finally, risk assessment studies consistently show the lack of increased significant risk of mesothelioma at exposure levels likely to be the result of exposure to chrysotile asbestos from work with, and in and around asbestos-containing floor covering (Hodgson et al., 2000; Berman and Crump, EPA, 2003). There has been a wide range of risk assessments comparing the relative potency between chrysotile's and amphibole's ability to produce mesothelioma. Barly research discussed estimated differences of several-fold. However more recent risk assessments note a much larger difference. Hodgson and Damton reported the following:

Crocidolite - 500 times more potent than chrysofile; Amosite - 100 times more potent than chrysofile. Berman and Crump in the 2003 draft EPA risk assessment conclude with a potency coefficient for chrysotile fibers that is 0.0013 times that for amphibole fibers.

There is significant scientific literature pointing towards a familial risk for mesothelioma (Ascoli et al, 1998; Huncharek et al, 1996; Ascoli et al, 2001; Hammar et al, 1989; Picklesimer et al, 2005; Bianchi et al, 2004; Risberg et al, 1980). However, certain genetic predisposition to mesothelioma development may make individuals significantly exposed to certain types of asbestos more likely to develop mesothelioma (Ohar et al, 2006; Bianchi et al, 1993; Heineman et al, 1996; Neri et al, 2005; Dawson et al, 1992).

## Alternative Factors

As noted above, there are other more likely etiologies for Mr. Nacht's mesothelioma, primarily, amphibole asbestos exposure. The scientific literature does demonstrate that various workers, who although not working directly with asbestos-containing products, such as electricians were exposed to asbestos and were at increased risk of developing asbestos related disease (Paik et al, 1983; Peto et al, 1995; Bovenzi et al, 1993; Subramanian and Madhavan,

Cynthia Weiss Antonucci, Esq. August 22, 2007

contain crocidolite and amosite as well as chrysotile asbestos. I am in agreement that the finding 2005; Morabia et al 1992; Hodgson et al, 1988; Guberan et al, 1989; Menck and Henderson, 1976; Dambar and Larssön, 1987). As noted above insulation products have been shown to of crocidolite in Mr. Macht's lungs along with the presence of pleural plaques would identify asbestos and specifically amphibole asbestos as the etiologic agent for Mr. Macht's mesothelioma.

#### SUMMARY

assessment of the current medical/scientific state of the art and my experience and training in the field of occupational and environmental medicine, it is my opinion that it is medically and ype and level necessary to impact the development or progression of his unfortunate disease. If indeed, asbestos-related, his mesothelioma is more likely than not the consequence of exposure mesothelioma, there is no reliable scientifically-derived evidence that his work selling asbestos projects, either residentially or commercially would produce any asbestos exposure of the fiber to amphibole fiber exposures which likely occurred during work around pipe insulation, boiler In summary, after careful review and evaluation of the case medical and other records, containing floor tile or supervising his employees installing and cleaning up floor installation scientifically more likely than not that while Mr. Nacht developed malignant pleural insulation and other amphibole asbestos from other sources.

The opportunity to provide this assessment is appreciated. If there are any questions regarding this report or if additional assistance is desired, please do not hesitate to call.

Sincerely,

Howard M. Sandler, M.D.

Occupational and Environmental Medicine

HMS/am

### REFERENCES

- Acheson E.D. et al. Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow up. British Journal of Industrial Medicine, 39:344-348, 1982.
- Agudo A. et al. Occupation and risk of malignant pleural mesothelioma: A case control study in Spain. American Journal of Industrial Medicine, 37(2):159-168, February 2000.
- Analysis of Measurements of Airborne Fibers During Removal of Resilient Floor Tiles Using Recommended Work Practices, ENVIRON Corporation, December 13, 1988.
- Antman K. et al. Update on malignant mesothelioma. Oncology, 19(10):1301-1309, September 2005.
- 5. Asbestos Risk Assessment, Epidemiology and Health Effects. Editors Dodson R.F. and Hammar S.P. Taylor & Francis, 2006.
- Ascoli V. et al. DNA copy number changes in familial malignant mesothelioma. Cancer Genet Cytogenet, 127(1):80-82, May 2001.
- Ascoli V. et al. Familial pleural malignant mesothelioma: clustering in three sisters and one cousin. Cancer Lett, 130(1-2):203-207, August 14, 1998.
- Bani-Hani K.E. and Gharaibeh K.A. Malignant peritoneal mesothelloma. J Surg Oncol, 91(1):17-25, July 1, 2005.
- Becklake M.R. et al. Fiber burden and asbestos-related lung disease: determinants of doseresponse relationships. American Journal of Respiratory and Critical Care Medicine, 150(6 Pt 1): 1488-92, December 1994.
- Berman D.W. and Crump K.S. Final Draft: Technical Support Document for a Protocol to. Assess Asbestos-Related Risk. U.S. Environmental Protection Agency, October 2003.
- Berry G. et al. Malignant pleural and peritoneal mesotheliomas in former miners and millers of erocidolite at Wittenoom, Western Australia. Occupational Environmental Medicine, 61(4):1-3, 2004.
- Bertino P. et al. Erionite and asbestos differently cause transformation of human mesothelial cells. Int J Cancer, March 12, 2007.

- Bianchi C. et al. Asbestos exposure in malignant mesothelioma of the pleura: a survey of 557 cases. Industrial Health, 39:161-167, 2001.
- Bianchi C. et al. Asbestos-related familial mesothelioma. Eur J Cancer Prev, 2(3):247-250; May 1903

14.

- 15. Bianchi C. et al. Familial mesothelioma of the pleura. Industrial Health, 42:235-239, 2004.
- Biological Effects of Asbestos. Whipple, H.E. Ed. The New York Academy of Sciences, December 31, 1965.
- Boffetta P. Epidemiology of peritoneal mesothelioma: a review. Ann Oncol, October 9, 2006.
- Boutin G, et al. Bilateral pleural plaques in Corsica: a marker of non-occupational asbestos exposure. IARC Sci Publ, 90:406-10, 1989.
- Bovenzi M. et al. Occupational exposure and lung cancer risk in a coastal area of northeastern Italy. Int Arch Occup Environ Health, 65(1):35-41, 1993.
- Brackett, K.A., Ph.D., Clark, P.J. and Powers, T.J. "TEM Observations of Airborne
  Asbestos Structures During the Removal of Vinyl Asbestos Tiles and Mastic Adhesive,"
  Environmental Choices Technical Supplement, July/August 15-20, 1992.
- Britton M. The epidemiology of mesothelioma. Seminars in Oncology, 29(1):18-25, 2002.
- Browne K, and Smither W.J. Asbestos-related mesothelioma: factors discriminating between pleural and peritoneal sites. Br J Ind Med, 40:145-152, 1983.
- Burdorf A. et al. Explaining differences in incidence rates of pleural mesothelioma between Sweden and the Netherlands. International Journal of Cancer, 113:298-301, 2005.
- Burnor K.J. et al. Expositre to brake dust and malignant mesothelioma: A study of 10 cases with Mineral Fiber Analyses. Annals of Occupational Hygiene, 47(4):325-330, 2003.
- Campopiano A., Casciari S., Fioravanti F., and Ramires D. Airborne asbestos levels in school buildings in Italy. Journal of Occupational and Environmental Hygiene, 1:256-261, 2004
- Chuang S.C. et al. Radiotherapy for primary thyroid cancer as a risk factor for second primary cancers. Cancer Lett, 238(1):42-52, July 8, 2006.

- 27. Churg A. Deposition and clearance of chrysotile asbestos. Annals of Occupational Hygiene, 38(4):625-633, 1994.
- Churg A. Fiber counting and analysis in the diagnosis of asbestos-related disease. Human Pathology, 13:381-392, 1982.
- Churg A. The distribution of amosite asbestos in the periphery of the normal human lung. British Journal of Industrial Medicine, 47:677-681, 1990.
- Churg A. and Stevens B. Absence of amosite asbestos in airway mucosa of non-smoking long term workers with occupational exposure to asbestos. British Journal of industrial Medicine, 50:355-359, 1993.
- Chung A. and Vedal S. Fiber burden and patterns of asbestos related disease in workers with heavy mixed amosite and chrysotile exposure. American Journal of Respiratory Critical Care, 150:663-669, 1994.
- Chung A. and Wiggs B. Fiber size and number in amphibole asbestos-induced mesothelioma. American Journal of Pathology, 115:437-442, 1984.
- Churg A. and Wiggs B. Mineral particles, mineral fibers, and lung cancer. Environmental Research, 37:364-372, 1985.
- Churg A. and Wright J.L., Persistence of natural mineral fibers in human lungs: An overview, Environmental Health Perspectives, 102(Suppl 5):229-233, 1994.
- Churg A. et al. Fiber size and number in workers exposed to processed chrysotile asbestos, chrysotile miners and the general population. American Journal of Industrial Medicine, 9:143-152, 1986.
  - Churg A. et al. Fibre content of lung in amphibole- and chrysotile-induced mesothelioms: implications for environmental exposure. IARC Sci Publ, 90:314-318, 1989.
    - Chung A. et al. Lung asbestos content in chrysotile workers with mesothelloma. American Review of Respiratory Disease, 130:1042-1045, 1984.
- 38. Churg A, et al. Environmental pleural plaques in residents of a Quebec chrysotile mining town. Chest, 94:58-60, 1988.
- Churg A, et al. Fiber burden and patterns of asbestos-related disease in chrysotile miners and millers. Am Rev Respir Dis, 148(1):25-31, July 1993.

- Churg A. Asbestos Fibers and Pleural Plaques in a General Autopsy Population. Am Assoc Pathol, 109(1):88-96, 1982.
- Cocco P. and Dosemeci M. Peritoneal cancer and occupational exposure to asbestos: results from the application of a job-exposure matrix. Am J Ind Med, 35(1):9-14, January 1999.
- Crossman R.N. et al. Quantification of fiber releases for various floor tile removal methods. Applied Occupational and Environmental Hygiene, 1113-1124, September 1996.
- Damber L.A. and Larsson L.G. Occupation and male lung cancer: a cass-control study in northern Sweden. Br J Ind Med, 44(7):446-453, July 1987.
- 44. Dawson A. et al. Familial mesothelioma. Cancer, 70:1183-1187, 1992.
- 45. Dement et al. Estimates of dose-response for respiratory cancer among chrysottle asbestos textile workers. Annals of Occupational Hygiene, 26(1-4):869-887, 1982.
- Dodson R.F. et al. Analysis of asbestos fibers burden in lung tissue from mesothelioma patients. Ultrastructural Pathology, 21:321-336, 1997.
- Dodson R.F. et al. Asbestos burden in cases of mesothelioma from individuals from various regions of the United States. Ultrastructural Pathology, 29:415-433, 2005.
- 48. Dodson R.F. et al. Asbestos content of omentum and mesentery in nonoccupationally exposed individuals. Toxicology and Industrial Health, 17:138-143, 2001.
- Dodson R.F. et al. Quantitative analysis of asbestos burden in women with mesothelioma.
   American Journal of Industrial Medicine, 43(2):188-195, February 2003.
- Dogan A.U. et al. Genetic predisposition to fiber careinogenesis causes a mesothelioma epidemic in Turkey. Cancer Research, 66(10):5063-5068, 2006.
- Duffesne A. et al. Fibers in lung tissues of mesothelioma cases among miners and millers
  of the township of Asbestos, Quebec. American Journal of Industrial Medicine, 27:581-592,
  1995.
- Duffesne A. et al. Mineral fiber content of lungs in patients with mesothelloma seeking compensation in Quebec. American Journal of Respiratory and Critical Care Medicine, 153(2):711-8, February 1996.

- 53. Duffesne A. et al. Retention of asbestos fibres in lungs of workers with asbestosis, asbestosis and lung cancer, and mesothelioma in asbestos township. Occupational and Environmental Medicine, 53(12):801-7, December 1996.
- Edwards, Alva, Kominsky, John R. and Freyberg, Ropald W. Airborne asbestos concentrations during spray-buffing of resilient floor tile," Applied Occupational and Environmental Hygiene, Yol. 9(2): 132-138, February 1994.
- Evaluation of Exposure to Airborne Fibers During Removal of Resilient Floor Tiles Using Recommended Work Practices, ENVIRON Corporation, April 3, 1989.
- Evaluation of Exposures to Airborne Fibers During Maintenance of Asbestos-Containing Resilient Floor Tiles Using Recommended Work Practices, ENVIRON Corporation, September 10, 1990.
- Evaluation of Worker Exposure to Airborne Fibers During the Removal of Resilient Floor Coverings and Asphaltic Cutback Adhesives Using Recommended Work Practices, BNVIRON Corporation, May 1, 1992.
- Final Report on Industrial Hygiene Survey of Asbestos Exposure During Resilient Floor
  Tile Removal Pursuant to Recommended Work Practices at 10 Resilient Floor Tile
  Removal Sites, Fowler Associates/Chatfield Technical Consulting, Ltd., December 1988.

. s

- Fischer M. et al. Fibre-years, pulmonary asbestos burden and asbestosis. International Journal of Hygiene and Environmental Health, 205(3):245-248. April 2002.
- Fonte R. et al. Asbestos-induced peritoneal mesothelloma in a construction worker. Environmental Health Perspectives, 112(5):616-619, 2004.
- GCA Corporation. Analysis of Fiber Release from Certain Asbestos Products, prepared for U.S. EPA Office of Pesticides and Toxic Substances Chemical Control Div., by GCA Corp., Bedford, MA, December 1982.
- Gibbs A.R. et al. Compatison of fibre types and size distributions in lung tissues of
  paraoccupational and occupational cases of malignant mesothelioma. British Journal of
  Industrial Medicine, 47:621-626, 1990.
- 63. Goldberg M. et al. The French National Mesothelloma Surveillance Program. Occup Environ Med, 63(6):390-395, June 2006.
- Goodman M. et al. Mesothelioma and lung cancer among motor vehicle mechanics: a meta-analysis. Annals of Occupational Hygiene, 48(4):309-326, 2004.

- Green P.H.Y. et al. Exposure and mineralogical correlates of pulmonary fibrosis in chrysotile asbestos workers. Occupational and Environmental Medicine, 54:549-559, 1997.
- Guberan E. et al. Disability, mortality and incidence of cancer among Geneva painters and electricians: a historical prospective study. Br J Ind Med, 46(1):16-23, January 1989.
- Gulmez I. et al. Evaluation of malignant mesothelioma in central Anatolia: a study of 67 cases. Can Respir J, 11(4);287-290, 2004.
- 68. Gylseth B. et al. Fibre type and concentration in the lungs of workers in an asbestos cement factory. British Journal of Industrial Medicine, 40:375-379, 1983.
- Hammar S.P. et al. Familial mesothelioma: a report of two families. Hum Pathol, 20(2):107-112, February 1989.
- Heineman B.F. et al. Mesothelioma, asbestos, and reported history of cancer in first-degree relatives. Cancer, 77(3):549-554, February 1, 1996.
- Henderson D.W. et al. The diagnosis and attribution of asbestos-related diseases in an Australian context: report of the Adelaide workshop on asbestos-related diseases, October 6-7, 2000. Int J Occup Environ Health, 10:40-46, 2004.
- 72. Hessel P.A. et al. Mesothelioma among brake mechanics: an expanded analysis of a case-control study. Risk Analysis, 24(3):547-552, 2004.
- 73. Hodgson J.T. et al. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. Annals of Occupational Hygiene, 44(8):565-601, 2000.
- 74. Hodgson M.J. et al. Asbestosis among electricians. J Occup Med, 30(8):638-649, August 1988.
- Hoekstra A.V. et al. Well-differentiated papillary mesothelloma of the peritoneum: a
  pathological analysis and review of the literature. Gyencol Oncol, 98(1):161-167, July 2005.
  - Howel D. et al. Mineral fibre analysis and routes of exposure to asbestos in the
    development of mesothelioma in an English region. Occupational Environmental Medicine,
    56:51-58, 1999.
- Hughes R.S. Malignant pleural mesothelioma. American Journal of Medical Science 329(1): 29-44, 2005.
- Huncharek M. et al. Parental cancer and genetic predisposition in malignant pleural mesothelioma: a case-conitol study. Cancer Lett, 102(1-2):205-208, April 19, 1996.

- 79. Huncharek M. et al. Pleural Mesothelioma in a brake mechanic. British Journal of Industrial Medicine, 46:69-71, 1989.
- Huncharek M. et al. Pleural Mesothelioma in a lift mechanic. British Journal of Industrial Medicine, 46:500-501, 1989.
- Jarvholm B. and Sanden A. Lung cancer and mesothelioma in the pleura and peritoneum among Swedish insulation workers. Occupational and Environmental Medicine, 55:766-770, 1998.
- Jaurand M.C. and Fleury-Feith J. Pathogenesis of malignant pleural mesothelioma. Respirology, 10(1):2-8, January 2005.
- Kominsky, John R., Freyberg, Ronald W. and Boiano, James M. "Airborne Asbestos Concentrations During Buffing, Burnishing, and Stripping of Resilient Floor Tile," BPA/600/SR-95/121, U.S. Environmental Protection Agency, August 1995.
- Laden F. et al. Lung cancér and mesothelioma among male automobile mechanics: a review. Reviews on Environmental Health, 19(1):39-61, 2004.
- Lange J.H. Impact of asbestos concentrations in floor tiles on exposure during removal. International Journal of Environmental Health Research, 12:293-300, 2002.
- Lange, J.H., Lange, P.R., Reinhard, T.K. and Thomulka, K.W. A study of personal and area airborne asbestos concentrations during asbestos abatement: A statistical evaluation of fibre concentration data. The Annals of Occupational Hygiene, 40(4):449-466, 1996.
- Langer A.M. et al. Asbestos in the lungs of persons exposed in the USA. Monaldi Arch Chest Dis, 53(2):168-180, 1998.
- Langer A.M. et al. Fibre type burden in parenchymal tissues of workers occupationally exposed to asbestos in the United States. IARC Sci Pupl, (90):330-5, 1989.
- Langer A.M. et al. Mesothelioma in a brake repair worker. Lancet, 2(8307):1101-1103,
   November 13, 1982.
- Leigh J. et al. Lung asbestos fiber content and mesothelioma cell type, site and survival. Cancer, 68:135-141, 1991.
- Li L. et al. Cohort studies on cancer mortality among workers exposed only to chrysotlic asbestos; a meta-analysis. Biomedical and Environmental Sciences, 17:459-468, 2004.

- Lin R.T. et al. Ecological association between asbestos-related diseases and historical
  asbestos consumption: an international analysis. Lancet, 369(9564):844-849, March 10,
  2007
- 93. Lippman M. Deposition and retention of inhaled fibres: effects on incidence of lung cancer and mesothelioma. Occupational and Environmental Medicine, 51:793-798, 1994.
- Lippman M. Effects of fiber characteristics on lung deposition, retention, and disease. Bnylronmental Health Perspectives, 88:311-317, 1990.
- Lundgren, Dale A., Vanderpool, Robert W. and Liu, Benjamin Y.H. Measurement of asbestos fiber concentration above floor tile. Particle & Particle Systems Characterization, Vol. 8:229-232, 1991.
- Lundgren, Dale A., Vanderpool, Robert W. and Liu, Benjamin Y.H. Additional measurement of asbestos fiber concentration above floor tile. October 1987.
- 97. Lundgren, Dale A., Vanderpool, Robert W. and Liu, Benjamin Y.H. Asbestos fiber concentrations resulting from the installation, maintenance and removal of vinyl-asbestos floor tile. January 1988.
- 98. Lundgren, Dale A., Vanderpool, Robert W. and Liu, Benjamin Y.H. Acrosol generation from the sanding, cutting, and breaking of floor tiles. Particle & Particle Systems Characterization, Vol. 7:121-126, 1990.
- Lundgren, Dale A., Vanderpool, Robert W. and Liu, Benjamin Y.H. Asbestos fiber concentrations resulting from the installation, maintenance and removal of vinyl-asbestos floor tile. Particle & Particle Systems Characterization, Vol. 3: 233-236, 1991.
- 100. Lundgren, Dale A., Vanderpool, Robert W. and Liu, Benjamin Y.H. Measurement of asbestos fiber concentration above floor tile. October 1987.
- 101. Manda-Stachouli C, et al. Decreaseing Provalence of Pleural Calfoifications Among Metsovites With Nonoccupational Asbestos Exposuré. Chest, 126:617-621, 2004.
- 102. Manzini V. Malignant peritoneal mesothelloma. Tumori, 91:1-5, 2005.
- 103. Marchevsky A.M. and Wiok M.R. Current controversies regarding the role of asbestos exposure in the causation of malignant mesothelioma: the need for an evidence-based approach to develop medicolegal guidelines. Annals of Diagnostic Pathology, 7(5):321-332, 2003.

- 104. Martonik J.F. et al. The history of OSHA's asbestos rulemakings and some distinctive approaches that they introduced for regulating occupational exposure to toxic substances. American Industrial Hygiene Association Journal, 62:208-217, March/April 2001.
- 105. McDonald A.D. et al. Malignant mesothelioma in Quebec. IARC Sci Publ, (30):673-80, 1980.
- 106. McDonald A.D. et al. Mesothelioma and fiber type in three American asbestos factories preliminary report. Scandinavian Journal of Work and Environmental Health, 8(Suppl 1):53-58, 1982.
- 107. McDonald A.D. et al. Mesothelioma in Quebec chrysotlic miners and millers: epidemiology and actiology. Annals of Occupational Hygiene, 41(6):707-719, 1997.
- 108. McDonald J.C. et al. Case-referent survey of young adult with Mesothelioma: I. Lung fibre analyses. Annals of Occupational Hygiene, 45(7):513-518, 2001.
  - 109. MoDonald J.C. et al. Chrysottie, tremolite and carcinogenicity. Annals of Occupational Hygiene, 41(6):699-705, 1997. Letter. Science 267(5199):776-777, February 10, 1995.
- 110. McDonald J.C. et al. Mesothelioma and asbestos fiber type. Evidence from lung tissue analyses. Cancer, 63(8):1544-1547, April 15, 1989.
- 111. McDonald J.C. et al. Sixty years on: the price of assembling military gas masks in 1940. Occupational Environmental Medicine, 63:852-855, 2006.
- 112. Meister T. et al. Papillary mesothelloma of the peritoneum in the absence of asbestos exposure. Z Gastroenterol, 41(4):329-332, April 2003.
- Menck H.R. and Henderson B.E. Occupational differences in rates of lung cancer. J Occup Med, 18(12):797-801, December 1976.
   Millette, J.R. and Ewing, E. Case Study: Drilling of Floor Tile During Residential Insect
  - Control. EIA Technical Monograph, Vol. 4:3-6, 1998.

    115. Morabia A. et al. Lung cancer and occupation: results of a multicentre case-control study.

    Br J Ind Med, 49(10):721-727, October 1992.
- 116. Mural Y. et al. Asbestos fiber analysis in 27 malignant mesothelioma cases. American Journal of Industrial Medicine, 22(2):193-207, 1992.
- 117. Murphy R.L. et al. Floor tile installation as a source of asbestos exposure. American Review of Respiratory Disease, 104:576-580, 1971.

- 118. Musti M. et al. A cluster of familial malignant mesothelioma with del(9p) as the sole chromosomal anomaly. Cancer Genetics and Cytogenetics, 138:73-76, 2002.
- 119. Nayebzadeh A. et al. Lung mineral fibers of former miners and millers from Thetford-mines and asbestos regions: A comparative study of fiber concentration and dimension. Archives of Environmental Health, 56(1):65-76, January/February 2001.
- 120. Neri M. et al. Pleural malignant mesothelioma, genetio susceptibility and asbestos exposure. Mutation Research, 2005.
- 121. Neugut A.I. et al. Incidence of malignant pleural mesothelioma after thoracic radiotherapy Cancer, 80(5):948-950, September 1, 1997.
- 122. Newhouse M.L. et al. A mortality study of workers manufacturing friction materials with chrysotile asbestos. Annals of Occupational Hygiene, 26(1-4):899-902, 1982.
- 123. Noian R.P. et al. Lung content analysis of cases occupationally exposed to ohrysottle asbestos. Environmental Health Perspectives, 102 Suppl 5:245-50, October 1994.
- March 2007.

124. Ohar J.A. et al. Identification of a mesothelioma phenotype. Respir Med, 101(3):503-509,

**₹** ₹

- 125. Paik N.W. et al. Worker exposure to asbestos during removal of sprayed material and renovation activity in buildings containing sprayed material. Am Ind Hyg Assoc J, 44(6):428-432, June 1983.
- 126. Paustenbach D.J., Sage A., Bono M., and Mowat R. Occupational exposure to airborne asbestos from coatings, mastics, and adhesives. Journal of Analysis and Environmental Epidemiology, 14:234-244, 2004.
- 127. Peto J. et al. Continuing increase in mesothelioma mortality in Britain. Lancet, 345(8949):535-539, March 4, 1995.
- 128. Picklesimer A.H. et al. Case report: malignant peritoneal mesothelioma in two siblings. Gynecologic Oncology, 99:512-516, 2005.
- 129, Pira E. et al. Cancer morfality in a cohort of asbestos textile workers. British Journal of Cancer, 92:580-586, 2005.
- 130. Removal of Non-Friable Asbestos-Containing Floor Tile Using the Resilient Floor Covering Manufacturer "Recommended Work Practices for the Removal of Resilient Floor Covering," Micro-Analytics, Inc., June 10, 1992.

- Resilient Floor Covering Institute. Recommended Work Practices for Removal of Resilient Floor Coverings, January 1998.
- 132. Review and Analysis of Studies That Monitored Fiber Exposures During Maintenance of Asbestos-Containing Resilient Floor Tiles, ENVIRON Corporation, November 30, 1990.
- 133. Review and Analysis of Studies That Monitored Fiber Exposures During Removal of Asbastos-Containing Resilient Floor Covering Materials, ENVIRON Corporation, November 30, 1990.
- 134. Risberg B. et al. Familial clustering of malignant mesothelioma. Cancer, 45:2422-2427, 1080
- 135. Robinson C. et al. Mortality patterns, 1940-1975 among workers employed in an asbestos textile friction and packing products manufacturing facility. Circa 1979.
- 136. Rogers A.J. et al. Relationship between lung asbestos fiber type and concentration and relative risk of Mesothelioma. Cancer, 67:1912-1920, 1991.
- 137. Roggli V.L. The role of analytical SEM in the determination of causation in malignant mesothelioma. Ultrastructural Pathology, 30:31-35, 2006.

**⊷** ≅

- 138. Roggli V.L. et al. Malignant mesothelioma and occupational exposure to asbestos: a clinicopathological correlation of 1445 cases. Ultrastructural Pathology, 26(2):55-65, Mar-Apr 2002.
- Roggli V.L. et al. Tremolite and mesothelloma. Annals of Occupational Hygiene, 46(5):447-453, 2002.
- 140. Rohl A.N. et al. Asbestos exposure during brake lining maintenance and repair. Environmental Research, 12(1):110-28, August 1976.
- 141. Rohl A.N. et al. Exposure to asbestos in the use of consumer spackling, patching, and taping compounds. Science, 189:551-553, August 1975.
- 142. Rubino G.F. et al. Mortality of chrysotile asbestos workers at the Balangero Mine, Northern Italy. British Journal of Industrial Medicine, 36(3):187-194, August 1979.
- 143. Sakai K. et al. Asbestos and nonasbestos fiber content in lung tissue of Japanese patients with malignant mesothelioma. Cancer, 73:1825-1835, 1994.
- 144. Sanders C.L. Pleural mesothelioms in the rat following exposure to 239PuO2. Health Phys, 61(6):695-697, December 1992.

- 145. Saracci R. and Simonato L. Familial malignant mesothelioma. Lancet, 358(9295):1813-1814, November 24, 2001.
- 146. Serio G. et al. Familial pleural mesothelioma with environmental asbestos exposure: losses of DNA sequences by comparative genomic hybridization (CGH). Histopathology, 45(6):643-645, December 2004.
- 147. Shilnikova N.S. et al. Cancer mortality risk among workers at the Mayak nuclear complex. Radiat Res, 159(6):787-798, June 2003.
- 148. Smith A.H. et al. Chrysotile asbestos is the main cause of pieural mesothelioma. American Journal of Industrial Medicine, 30:252-266, 1996.
- 149. Spirtas R. et al. Malignant Mesothelioma: attributable risk of asbestos exposure. Occupational and Environmental Medicine, 51:804-811, 1994.
- Spirtas R. et al. Malignant mesothelioma: attributable risk of asbestos exposure. Occup Environ Med, 51(12):804-811, December 1994.
- 151. Spirtas R. et al. Mesothelioma risk related to occupational or other asbestos exposure: preliminary results from a case control study. Society for Epidemiologic Research: Abstracts. American Journal of Epidemiology, 122:518, 1985.

F 5

- 152. Srebro S.H. et al. Asbestos-related disease associated with exposure to asbestiform Tremolite. American Journal of Industrial Medicine, 26:809-819, 1994.
- 153. SRI International. Comparison Testing Monitoring for Airborne Asbestos Fibers: Sheet Vinyl Floor Covering, Wet Versus Dry Scraping, Final Report, SRI International Project 7988, November 1979.
- 154. SRI International. Monitoring for Airborne Asbestos Fibers: Vinyl Asbestos Floor Tile, Final Report, SRI International Project 7988, December 1979.
- 155. Stanton M.F. and Wrench C. Mechanisms of mesothelioma induction with asbestos and fibrous glass. Journal of the National Cancer Institute, 48:797-821, 1972.
  156. Subramanian V. and Madhavan N. Asbestos problem in India. Lung Cancer, 49 Suppl
  - 1:89-S12, July 2005.
- 157. Suzuki Y. et al. Asbestos fibers contributing to the induction of human malignant mesothelioma. Annals of the New York Academy of Science, 982:160-76, December 2002.

Cynthia Weiss Antonucci, Esq. August 22, 2007

- 158. Suzuki Y. et al. Asbestos tissue burden study on human malignant mesothelioma. Industrial Health, 39(2):150-160, April 2001.
- [59. Svorcan P. et al. Primary malignant mesothelioma of the peritoneum. Rom J Gastroenterol, 12(2):135-137, June 2003.
- 160. Teschke K. et al. Mesothelioma surveillance to locate sources of exposure to asbestos. Canadian Journal of Public Health, 88(3):163-168, May-June 1997.
- 161. Teta M.J. et al. Mesothelioma in Connecticut, 1955-1977. Journal of Occupational Medicine, 25(10):749-756, October 1983.
- 162. Thomas H.F. et al. Further follow up study of workers from an asbestos factory. British Journal of Industrial Medicine, 39:273-276, 1982.
- Tossavainen A. et al. Amphibole fibres in Chinese chrysotile asbestos. Annals of Occupational Hygiene, 45(2):145-52, March 2001.
- 164. Tossavainen A. et al. Pulmonary mineral fibers after occupational and Environmental Exposure to Asbestos in the Russian Chrysotlie Industry. American Journal of Industrial Medicine, 37:327-333, 2000.

e e

, ,

- 165. U.S. Bavizonmental Protection Agency. Asbestos-in-Buildings Technical Bulletin: Use of Asbestos-Containing Friable Materials and Vinyl-Asbestos Floor Tiles in Public and Commercial Buildings, U.S. Environmental Protection Agency, TS-798:1-4, 1984.
- 166. Ulvestad B. et al. Cancer incidence among workers in the asbestos-cement producing industry in Norway. Scand J Work Erviron Health, 28(6):411-417, 2002.
- 167. Vinyl Asbestos Floor Tile-Study, Routine Buffing and Stripping Operations for WRC-TV Washington, A.F. Meyer and Associates, Inc., November 6, 1989.
- 168. Wagner J.C. et al. Correlation between lung fibre content and disease in Bast London asbestos factory workers. IARC Sci Pubi, (90):444-448, 1989.
- 169. Wagner J.C. et al. Diffuse pleural mesothelloma and asbestos exposure in the North Western Cape Province. British Journal of Industrial Medicine, 17:260-271, 1960.
- 170. Wannock M.L. Lung asbestos burden in shipyard and construction workers with mesothelioma: comparison and burdens in subjects with asbestosis or lung cancer. Environmental Research, 50(1);68-85, October 1989.

- Warnock M.L. et al. Numbers and Types of Asbestos Fibers in Subjects With Pleural Plaques. Am Assoc Pathol, 109(1):37-46, 1982.
- 172. Weiss et al. Mortality of a cohort exposed to chrysotile asbestos. Journal of Occupational Medicine, 19(11);737-740, November 1977.
- 173. Weich L.S. et al. Asbestos and peritoneal mesothelioma among college-educated men. Int J Occup Environ Health, 11:254-258, 2005.
- 174. Williams M.G. and Crossman R.N. Asbestos release during removal of resilient floor covering materials by recommended work practices of the Resilient Floor Covering Institute. Applied Occupational and Environmental Hygiene, 18(6):466-478, 2003.
- Woitowitz HJ. et al. Mesothelioma among car mechanics, Annals of Occupational Hygiene, 38(4):635-638, 1994.
- 176. Wong I. Malignant mesothelioma and asbestos exposure among auto mechanics: appraisal of scientific evidence. Regulatory Toxicology and Pharmacology, 34:170-177, 2001.
- 177. Yan T.D. et al. Significance of lymph node metastasis in patients with diffuse malignant peritoneal mesothelioma. Eur J Surg Oncol, 32(9):948-953, November 2006.
- 178. Yano B. et al. Cancer mortality among workers exposed to amphibole-free chrysotile asbestos. American Journal of Epidemiology, 154(6):538-543, 2001.
- 179. Yarborough C.M. Chrysotile as a cause of mesothelioma; an assessment based on epidemiology. Critical Reviews in Toxicology, 36:165-187, 2006.

ALL-STATE LEGAL<sup>®</sup> 07181-BF - 07182-BL - 07183-GY - 07184-WH ... 630.222.0510 www.aslegal.com

Index No.

115546

Year 20 06

## SUPREME COURT OF THE STATE OF NEW YORK COUNTY OF NEW YORK

IN RE: NEW YORK CITY ASBESTOS LITIGATION

This Document Relates To:

FRANK BIANCO, et al.,

Plaintiffs,

- against -

A.O. SMITH WATER PRODUCTS, et al.,

Defendants.

SUPPLEMENTAL AFFIRMATION: IN SUPPORT OF PLAINTIFFS' MOTION, PURSUANT TO FRYE V.

UNITED STATES, AND IN LIMINE, 1) TO PRECLUDE THE NOVEL, UNSCIENTIFIC, NOT GENERALLY-ACCEPTED,
LITIGATION-BASED, PREVIOUSLY EXCLUDED ASBESTOS "DOSE RECONSTRUCTION"?"EXPOSURE ASSESSMENT"
ANALYSES AND ALL TESTIMONY RELATED THERETO, AND 2) TO PRECLUDE THE CASE-SPECIFIC REPORTS AND
RELATED TESTIMONY OF DRS. RABINOVITZ AND WEINBERG SPECIFICALLY AS IMPROPER

WEITZ & LUXENBERG, P.C.

Attorneys for

Plaintiffs .

180 Maiden Lane New York, NY 10038 (212) 558-5500

State, certif	22 NYCRR 130-1.1, t fies that, upon informa cument are not frivolous	ation and belief and i	torney admitte reasonable inq	d to practice in the courts quiry, the contentions con	s of New York tained in th
Dated:		Signature		***************************************	
		Print Signer's Na	me		
Service of a copy of the within			is hereby admitted		
Dated:					
		Attor	ney(s) for		
PLEASE T	AKE NOTICE				
NOTICE OF ENTRY	that the within is a (certified) true copy of a entered in the office of the clerk of the within named Court on				20
NOTICE OF SETTLEMENT	•				
	on	20	, at	<b>M</b> .	
Dated:					

WEITZ & LUXENBERG, P.C.

Attorneys for

180 Maiden Lane New York, NY 10038